VISUAL FUNCTION FOLLOWING OPTIC NEURITIS TREATMENT

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CHENNAI

CERTIFICATE

This is to certify that this dissertation entitled **"VISUAL FUNCTION FOLLOWING OPTIC NEURITIS TREATMENT"** is a bonafide work done by **Dr.D.ABIRAMASUNDARI** under our guidance and supervision in the Neuro-ophthalmology Department of Aravind Eye Hospital and Post Graduate Institute of Ophthalmology, Madurai during the period of her post graduate training in Ophthalmology for May 2012 - April 2015.

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DECLARATION

I, **Dr.D.ABIRAMASUNDARI**, hereby declare that this dissertation entitled, "**VISUAL FUNCTION FOLLOWING OPTIC NEURITIS TREATMENT".**

Is being submitted in partial fulfilment for the award of M.S.in Ophthalmology Degree by the Tamil Nadu DR.MGR Medical University in the examination to be held in April 2015.

I declare that this dissertation is my original work and has not formed the basis for the award of any other degree or diploma awarded to me previously.

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"Live as if you were to die tomorrow and learn as if you were to live forever"

"Feeling gratitude and not expressing it is like wrapping a present and not giving it"

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PART-I

PART-II

ABSTRACT

VISUAL FUNCTION FOLLOWING OPTIC NEURITIS TREATMENT

AIM:

To evaluate visual function following optic neuritis treatment in South India.

MATERIALS AND METHODS:

This is a prospective study carried out over a period of 13 months , 98 eyes were examined and followed up for 3 months. Visual function following treatment was assessed.

RESULTS:

Mean age was 40.0 years. Male preponderance was seen (53.4%). Papillitis (65.3%) was more common than retrobulbar neuritis (34.7%). Bilateral presentation was seen in 20.3%. Baseline median logMAR visual acuity was 1.48 which improved to 0.3 a t 1 month follow up. Approximately 78% of eyes showed significant improvement in visual acuity. Other visual function including colour vision, central fields, Brightness sensitivity and red desaturation showed statistically significant improvement following treatment. Only 3 patients in our study showed signs of associated demyelination.

CONCLUSION:

Visual function following treatment showed a good recovery. There was less incidence of associated demyelinating disease in our study.

KEYWORDS:

Optic neuritis, visual acuity, central fields, dyschromatopsia, brightness sensitivity, red desaturation, demyelination.

INTRODUCTION

Optic neuritis is an infective, inflammatory or demyelinating disease affecting the optic nerve. It is characterised by sudden loss of vision often accompanied by pain which can be lasting for several hours or days followed by gradual recovery. Women are more commonly affected than men than men. Most of the cases are idiopathic and it can also be associated with multiple sclerosis. It is the most common demyelinating disease causing optic neuritis. Other common causes include infectious, parainfectious , inflammatory, paravaccination and immunological responses.

Optic neuritis can be typical (associated with multiple sclerosis) presents as acute uniocular vision loss often associated with pain that worsens on eye movements. RAPD is present in almost all unilateral cases. Variety of field defects and dyschromatopsia is noted in affected eyes. Recovery of vision usually begins within the first month. It improves independent of steroids. Atypical optic neuritis includes absence of pain , marked swelling of optic nerve with retinal exudates and haemorrhages. Patients with atypical features of neuritis are at lower risk of developing multiple sclerosis.

An episode of optic neuritis increases the risk of developing multiple sclerosis. Optic neuritis is referred to as Mono symptomatic or Idiopathic or as a Clinically Isolated Syndrome in the absence of signs of multiple sclerosis.

The clinical triad of optic neuritis consist of

1. Visual loss
2. Periocular pain
3. Dyschromatopsia.

Decreased vision associated with pain on eye movements is most common in retrobulbar optic neuritis. Pain may occur due to traction of origins of the medial and superior recti on optic nerve sheath at the orbital apex. (Whitnall's hypothesis) [1.]

Other aspects of visual function such as colour vision, contrast sensitivity, and visual field are affected. Visual field defects are variable in patients with optic neuritis which ranges from diffuse depression, centrocaecal scotoma to quadrantic defects and altitudinal defects. Optic neuritis is not recovered completely.[2,47]

Rapid recovery of vision occurs with the use of corticosteroids and it is the main stay of treatment. There are reports that even after six to twelve months there is not much difference in visual outcome. A pulse therapy of intravenous methyl prednisolone in a dose of 500mg twice a day was given for 3 consecutive days followed by oral steroids of 1mg / kilogram body weight was given for 11 days is given.

The gold standard treatment for Optic neuritis is based on Optic Neuritis Treatment Trial. It was undertaken to determine the efficacy of corticosteroids which enrolled 455 patients between 1988 and 1991. ONTT evaluated the efficacy of corticosteroid treatment for acute optic neuritis and relationship between optic neuritis and multiple sclerosis.

REVIEW OF LITERATURE

HISTORY:

Optic neuritis was first described by Nettleship in 1884. Gunn in 1891 defined optic neuritis as rapid failure of visual acuity usually more marked in one eye. Buning et al indicated that there is no correlation between initial visual acuity or treatment and residual visual function disturbances. Uthoff 1904 described a component of interstitial inflammation in the connective tissue septa along with demyelination of optic nerve. [2,3,4]

Morphologically it is classified into

1. Papillitis – associated with swollen optic disc .
2. Retrobulbar neuritis - disc appears normal.
3. Neuroretinitis – inflammation of nerve fibre layer and macular star in late stages.

   Isolated optic neuritis is referred as with absence signs of multiple sclerosis or any systemic diseases.

Severity of vision loss can vary from slight deficit to complete loss of light perception. Even if the visual recovery is complete few may have abnormalities found in contrast sensitivity, colour vision, stereopsis and optic disc appearance.[1]

ON can occur in isolation or in association with multiple sclerosis.[5] Women are most commonly affected than men.

The gold standard treatment for optic neuritis is based on the Optic Neuritis Treatment Trial which was to determine the efficacy of corticosteroids and to permit long term analysis.

ANATOMY OF OPTIC NERVE

It is a second cranial nerve. It extends from optic disc and upto optic chiasma. It is the backward continuation of the nerve fibre layer of the retina which consist of axons originating from ganglion cells. It also contains light reflex fibres and some centrifugal fibres. It is surrounded by meninges.

The optic nerve fibres are about million, 2-10 microns in diameter. It is about fifty millimeters in length.

- It is divided into four parts
- Intraocular part (1mm)
- Intraorbitalpart (30mm)
- Intracanalicularpart (6-9mm)
- Intracranial part (10mm)

INTRAOCULAR PART

Optic nerve pass through sclera, choroid and finally appears in the eye as optic disc. The average diameter of this part is 1.5mm, it expands 3mm behind the sclera where the neurons acquire a myelin sheath. It is divided into four layers from anterior to posterior.

1. **Surface nerve fibre layer:**

   It is essentially composed of axonal bundles i.e nerve fibres of retina(94%)which converge on optic disc and astrocytes(5%). The optic disc covered by thin layer of astrocytes.the internal limiting membrane of Elschnig, which seperates it from vitreous and is continuous with the internal limiting membrane of retina. The central portion of this membrane thickened known as centfral meniscus of Kuhnt. All the layers of retina apart from nerve fibre layer near the optic nerve are separated by partial rim of glial tissue called intermediate tissue of Kuhnt.

2. **Prelaminar region:**

   The predominant structure at this level are neurons and significantly increased quantity seperates of astroglial tissue. The border tissue of Jcoby seperates the nerve from choroid.

3. **Lamina cribrosa:**

   It is a fibrillar sieve like structure made up of fenestrated sheets of scleral connective tissue lined by glial tissue. It bridges the scleral canal. The bundle of optic nerve fibres leave the through these fenestrations. The border tissue of Elschnig intervenes between the choroid and sclera and optic nerve fibres. It gets its rich blood supply from Circle of Zinn.

4. **Retrolaminar region:**

   It is characterised by a decrease in astrocytes and acquisition of myelin that is supplied by oligodendrocytes. The addition of myelin sheath doubles the diameter of optic nerve.

INTRAORBITAL PART:

It starts from eyeball to optic foramen. It is covered by dura, arachnoid, pia. The pial sheath contains capillaries and sends septae to divide the nerve into fasciculi. The subarachnoid space contains cerebrospinal fluid. The central retinal artery along with its vein crosses the subarachnoid space to enter the nerve on its inferomedial part. Anteriorly nerve is separated from extraocular muscles by the orbital fat. Posteriorly optic nerve is surrounded by annulus of Zinn and orgin of four rectus muscles. Some fibres of superior rectus muscle and medial rectus muscle are adhered to optic nerve sheath and accounts for pain on ocular movements seen in retrobulbar neuritis. The long and short ciliary nerves and arteries surround the optic nerve before these enter the eyeball.

INTRACANALICULAR PART:

It is in close relation to ophthalmic artery where the nerve crosses inferiorly from medial to lateral side in the dural sheath and then leaves the sheath at the orbital end of the canal. Medially sphenoid and posterior ethmoidal sinuses are separated by a thin bony lamina.

INTRACRANIAL PART:

In this part it is about 1 cm in length. It lies above the cavernous sinus where the two nerves meet to form the chiasma. It is ensheathed in piamater but receives arachnoid and dural sheaths at the point of its entry into optic canal.

ARRANGEMENT OF NERVE FIBRES:

1. In the optic nerve head: Peripheral retinal fibres lie deep in retina. It occupies the superficial part of optic disc. Fibres which are present close to the ON head lie superficially in the retina and occupies more deep portion of the disc.

THICKNESS OF NFL AT THE DISC:

It progressively increases around different quadrants of optic disc margin

- Most lateral quadrant (thinnest)
- Upper temporal and lower temporal quadrant
- Most medial quadrant
- Upper nasal and lower nasal quadrant(thickest)

2. In the distal region: The upper temporal and lower temporal fibres are situated on the temporal half of the optic nerve and are separated from each other by a wedge shaped area occupied by the papillomacular bundle.
3. In the proximal region:

   The macular fibres are centrally placed.

VASCULAR SUPPLY OF OPTIC NERVE:

1. **Optic nerve head:**
   a. Surface nerve fibre layer: Its mainly supplied by capillaries derived from retinal arterioles. It anastomose with vessels of prelaminar region. Occasionally ciliary derived vessel from prelaminar region may enlarge to form cilioretinal artery.
   b. The prelaminar region: It is primarly derived from peripapillary choroidal system or separate branches of short posterior ciliary arteries.
   c. The lamina cribrosa region: It is supplied by ciliary vessels which are derived from short posterior ciliary arteries and arterial circle of Zinn –Haller.

d. The retrolaminar region: It is supplied by both ciliary and retinal circulation. The central retinal artery provides centripetal branches where it arisesfrom pial plexus and also centrifugal branches.

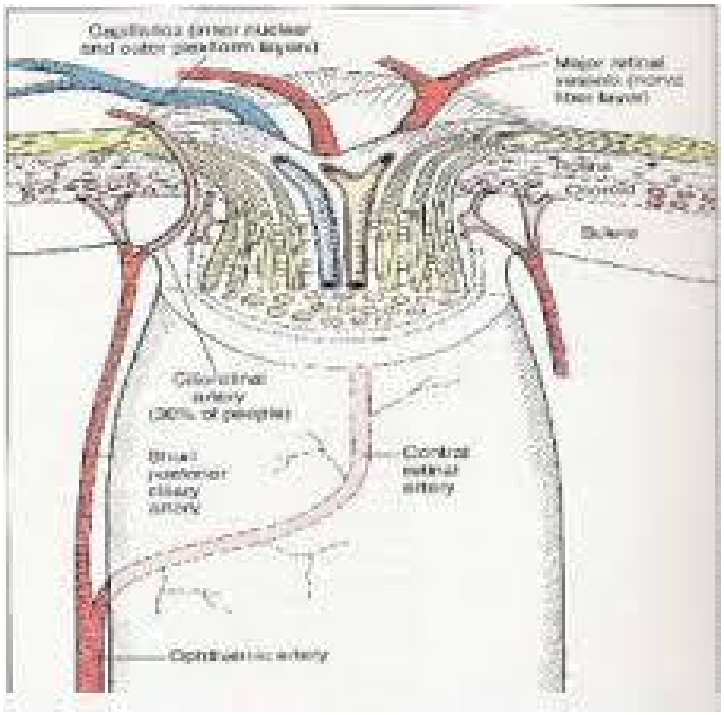


Fig 1 Blood Supply Of Optic Nerve Head

2. **The intraorbitalpart :** Its supplied by two system of vessels.
    a. **Periaxial :** It is derived from six branches of internal carotid artery.
        1. Ophthalmic artery
        2. Long posterior ciliary arteries
        3. Short posterior ciliary arteries

4. Lacrimal artery
5. Central artery of retina

b. **Axial system:** It is derived from
    1. Intra neural branches of central retinal artery.
    2. Central collateral arteries which comes from central retinal artery.
    3. Central artery of optic nerve.

3. The inracanalicular part:

It is supplied only by periaxial system of vessels. The pial plexus in this part is mainly fed from branches of ophthalmic artery.

4. The intracranial part:

It is exclusively supplied by periaxial system of vessels. The pial plexus is contributed by 4 sources

1. Branches from internal carotid artery directly or through recurrent branch of anterior hypophsealartey
2. Branches from anterior cerebral artery
3. Small recurrent branches from ophthalmic artery
4. Twigs from anterior communicating artery.

DEMOGRAPHY:

Most of the following information of visual outcome in optic neuritis comes from ONTT/LONS. The annual incidence of optic neuritis has been estimated in population based studies to be 1-5 per 1000,000.[6] Most patients are between ages of 20-50 years. Females are commonly affected than males. In ONTT, 77% were female, 85% were Caucasian and the mean age was 32±7 years.[5] . Optic neuritis can occur at any age. In Asia, ON is more common in relation to the incidence of MS in the United States of America or Western Europe.[8]

CLASSIFICATION:

Optic neuritis is classified into 4 types based on site of involvement

1. Retrobulbar neuritis – normal appearance of disc.
2. Papillitis – swollen optic disc
3. Perineuritis- its involvement of optic nerve sheath.
4. Neuroretinitis - papillitis associated with inflammation of nerve fibre layer and macular star in late stages.

Fig 1. Papillitis – marked inflammation of the optic disc

Fig 2 Retrobulbar neuritis- normal looking disc.

Fig 3 Neuroretinitis- marked inflammation around the optic disc with involvement of adjoining retina

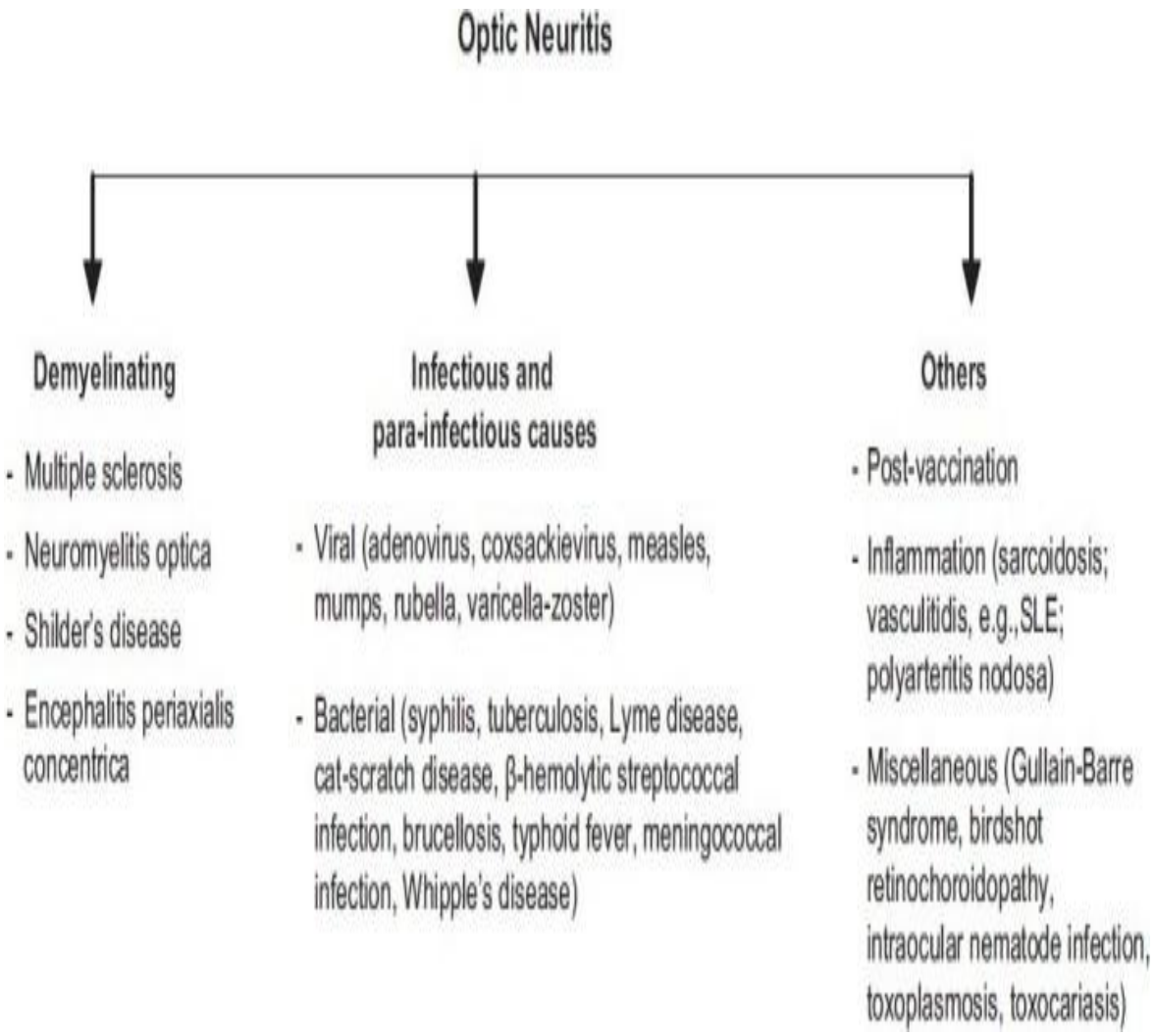
Based on topography, three principle type of neuritis is classified into

1) Perineuritis or periaxial neuritis
2) Axial neuritis
3) Transverse neuritis

ETIOLOGY

It is idiopathic in majority of cases. It could be associated with demyelinating lesion, of which most common cause is multiple sclerosis.

Other diseases known to cause optic neuritis are:



A) Viral infections (e.g. chicken pox, measles, mumps etc.)

B) Granulomatous infections such as tuberculosis, cat scratch disease, syphilis, Lyme disease or Cryptococcal meningitis.

C) Autoimmune diseases like SLE, Wegner ’s Granulamatosis or sarcoidosis

D) Contagious inflammation from orbit, meninges and sinuses

E) Intraocular inflammation involving retina, uvea, sclera.

MULTIPLE SCLEROSIS AND OPTIC NEURITIS

Optic neuritis is the most common initial manifestation of multiple sclerosis. Most common cause of visual disability among young and middle aged adults. MS is an acquired, chronic inflammatory disease of central nervous system. It is characterised by focal demyelinating lesions of varying age. It is reported to be most common aetiology in Western literature.[8,9] Incidence of MS in India and other Asian countries is low.[10,11,12]

After an attack of optic neuritis, possibility of developing MS reaches 38% in following 10 years and the percentage rises to 56% if there are brain abnormalities in MRI.[13.]

The Optic Neuritis treatment trial (ONTT) has shown female gender , one or more brain lesions on MRI, history of nonspecific neurological symptoms(usually transient numbness) , prior optic neuritis in fellow eye or retrobulbar neuritis are positive risk factors for development of Multiple Sclerosis. [14] Similarly negative risk factors are male gender, no lesions on MRI, optic disc swelling, absence of pain and ophthalmoscopic findings of severe optic disc edema, peripapillary haemorrhages or retinal exudates.

PATHOPHYSIOLOGY

Inflammatory demyelination in central nervous system including optic nerves and visual pathway. It was previously thought as disease of myelin with sparing of nerve axons however neuronal and axonal loss are known to occur in MS, leading to permanent neurologic and visual impairment.

Activated peripheral T cells migrate across the blood brain barrier and release cytokines and other inflammatory mediators leading to neuronal cell death and axonal degeneration. After an acute event axons get damaged which leads to loss of axons which cause severe and irreversible neurological impairment.

Diagnostic criteria for MS was based on Poser clinical criteria

1. Clinically definite MS
2. Two attacks (relapses) of > 24 hours duration and more than one month apart together with clinical evidence of lesion in two places within the central nervous system.

1. Laboratory supported definite MS includes evidence from lumbar puncture showing oligoclonal banding.

2. Clinically propable MS above combination of clinical and paraclinical evidence but no oligoclonal bands.
3. Laboratory supported propable MS oligoclonal banding without clinical or paraclinical evidence of lesions.[15]

Retrobulbar neuritis and papillitis are mainly associated with MS.

Perineuritis and neuroretinitis are most often associated with infectious or inflammatory pathologies. [16, 17,18, 19,13.] Neuroimaging (MRI) demonstrates white matter T2 signal abnormalities consistent with demyelination.[20]

Optic neuritis can be typical or atypical.[2,3,4,14,21] It is classified based on their clinical features and course of disease.

NEUROMYELITIS OPTICA:

It is variant of multiple sclerosis. Also known as Devics disease. It is an inflammatory or demyelinating disease of CNS. Patients can present with ON or transverse myelitis. Patients can present with unilateral or bilateral ON, transverse myelitis or both in close relationship to each other.

Features of NMO are visual loss caused by damage to the anterior visual sensory pathways and paraplegia caused by damage to the spinal cord. Pain is not a definite feature. No definite pattern of visual field loss is noticed. Fundus shows a mild disc swelling associated with venous dilatation and extensive peripapillary exudation. Optic atrophy sets as a final sequela. CSF examination reveals lymphocytic pleocytosis. MRI shows abnormal T2 weighed signals and enhancement with gadolinium in the optic nerves, chiasm and spinal cord.

INFECTIOUS/ PARAINFECTIOUS ON:

Direct infection of the nerve (viral or bacterial) or inflammation triggered by an immune reaction to systemic or central nervous infection may cause an optic neuritis. Viruses such as adenovirus, Coxsackie virus, CMV, Hepatitis A virus, EBV, HIV and Measles, Mumps, Rubella and Varicella Zoster viruses. Bacterial infections resulting in the form of inflammation include syphilis, Lymes disease , cat scratch disease. Optic neuritis usually occur within 1 to 3 weeks of the basic infection. This type of optic neuritis is more common in children. Optic disc may appear normal or may be swollen with associated peripapillary retinal edema. It may be associated with meningitis, encephalitis, or encephalomyelitis.

CSF may show pleomorphic lymphocytosis and an elevated protein concentration. Visual recovery is excellent without treatment. Corticosteroids hastens recovery.[2,3,4]

POST VACCINATION OPTIC NEURITIS :

ON can occur after vaccination against both bacterial and viral infections. It may develop after vaccination with BCG, Hepatitis B virus, Rabies virus, Tetanus Toxoid, DPT and MMR. Influenza vaccine is most commonly associated with such presentation. Onset usually within 1 to 3 weeks. Most commonly bilateral. Visual recovery occurs few weeks to months.[22,23,24]

Optic neuritis in multiple sclerosis is characterised by subacute / acute unilateral visual loss which stabilizes by 1 to 2 weeks in patients between age of 20 to 50 years with clinical and neurological evidence of multiple sclerosis and no evidence of other disease process.[2,3,4,21,9] .

Features of typical optic neuritis:

- Females are predominantly affected.
- F:M ratio is 5:1.

- Age 15 to 45 years
- Unilateral
- Acute and often painful loss of vision over hours to days.
- Pain on eye movements.
- Peak visual loss within 2 weeks.
- Visual loss is subtle to complete which starts improving thereafter.
- Relative afferent papillary defect often present.
- Poor colour vision and contrast sensitivity.
- Disc edema, vitreous cells, haemorrhages and cotton wool spots rarely present.
- Fundus appears normal in retrobulbar neuritis.

Any type of visual field defect may be seen but generalised depression of visual field is the most common presentation.

VER (Visually evoked response) shows prolonged latency with normal or depressed amplitude.

Patients may complain of residual deficits in contrast sensitivity, colour vision, stereopsis, light brightness, visual acuity or visual field.

Features of Atypical optic neuritis.

- Age less than 20 or greater than 50 years
- Painless loss of vision
- Persistent pain lasting for more than 7 days.
- Older patients are most commonly affected.
- Bilateral presentation
- Disc haemorrhages and cotton wool spots can occur.
- Lack of significant improvement of visual function or worsening within the first three weeks after onset of symptoms.
- Progression of visual field loss beyond 2 weeks.
- Diagnosis or evidence of other systemic conditions (inflammatory or infectious diseases including HIV infection) other than multiple sclerosis might cause optic neuropathy.
- Patients fail to improve with treatment.

CLINICAL ASSESMENT

1. History
2. Visual acuity
3. Colour vision
4. Pupil reflexes
5. Biomicroscopy and Fundoscopy.
6. Visual fields

Associated symptoms might be movement phosphenes, sound induced phosphenes, visual obscuration of bright light and Uhthoff ‘s phenomenon.

Other symptoms are less common. Loss of central vision is reported in 90% of patients.[9] Loss of vision is usually abrupt occurring several hours to days. In some cases its minimally reduced,there is complete blindness with no perception of light. Not all the patients complain of loss of central vision. Some complain loss of peripheral vision such as superior or inferior region,such patients deny loss of central acuity and may found to have 20/20 or better in the affected eye.[25] Pain in and around the eye is present in 90% of patients. It is usually mild but it may be severe and extremely debilitating than loss of vision.

1. Loss of vision

Loss of central visual acuity is the major symptom in most cases of acute optic neuritis being reported by over 90% of patients.[9] Loss of vision is abrupt occurring several hours to several days. The degree of visual loss varies widely. In some cases visual acuity is minimally reduced in others there is complete blindness with no perception of light. Visual loss is monocular in most cases, it can also be binocular. Best corrected visual acuity was measured with a wall mounted Snellens Visual acuity chart at 6 metres distance.

2. Loss of visual field

Few patients complains of loss of central field of vision, some complain of loss of peripheral vision usually in a particular area of visual field such as superior or inferior region. Such patients deny loss of central acuity and may found to have 6/6 vision in the affected eye.[25]

3. Ocular or orbital pain

Pain in and around the eye is present in more than 90% of patients. It is usually mild but it may be extremely severe and more debilitating to the patient than loss of vision. It may precede or concurrently occur with vision loss.

In ONTT pain was reported by 92% of patients of whom 87% indicated that it was worsened by eye movement. Rose theorized that the pain is caused by inflammation or swelling in the optic nerve sheath that are innervated by small branches of the trigeminal nerve.[26] Lepore reported that among 101 eyes with optic neuritis, pain was more commonly present with retrobulbar neuritis than with papillitis.[1] In ONTT pain was present in 93% of 295 eyes with retrobulbar neuritis and in 90% of 162 eyes with papillitis. Majid et al reported painful eye movements in 12 patients in his study.[51]

4. Positive visual phenomena

Patients with optic neuritis experience positive visual phenomena called photopsia in addition to pain and blurring both at onset of visual symptoms. Visual phenomena is precipitated by eye movements. Positive visual phenomena are reported by 30% of patients in the ONTT.

OTHER SYMPTOMS:

1) Movement phosphenes:

It can occur before an attack of optic neuritis or may accompany visual loss during the attack or may occur 6 months after full recovery. It especially occurs in horizontal movements in dim lit room; very brief flashes of light lasting only for 1 or 2 seconds occurring unilaterally and in ipsilateral affected eye even when it is maintained in lateral gaze. It is due to demyelination and demyelinated nerve fibres may discharge spontaneously when subjected to minimal mechanical stress.

2) Sound induced phosphenes:

Phosphenes precipitated by sudden noise.

3) Visual obscuration in bright light:

It is due to fluctuating interference in the transmission of visual signals.

4) Uhthoff's symptom:

It is an episodic transient obscuration of vision with exertion, hot baths, and showers. Patients have blurring of vision 5 to 20 minutes after exposure to provoking factor.

SIGNS:

1) Visual acuity:

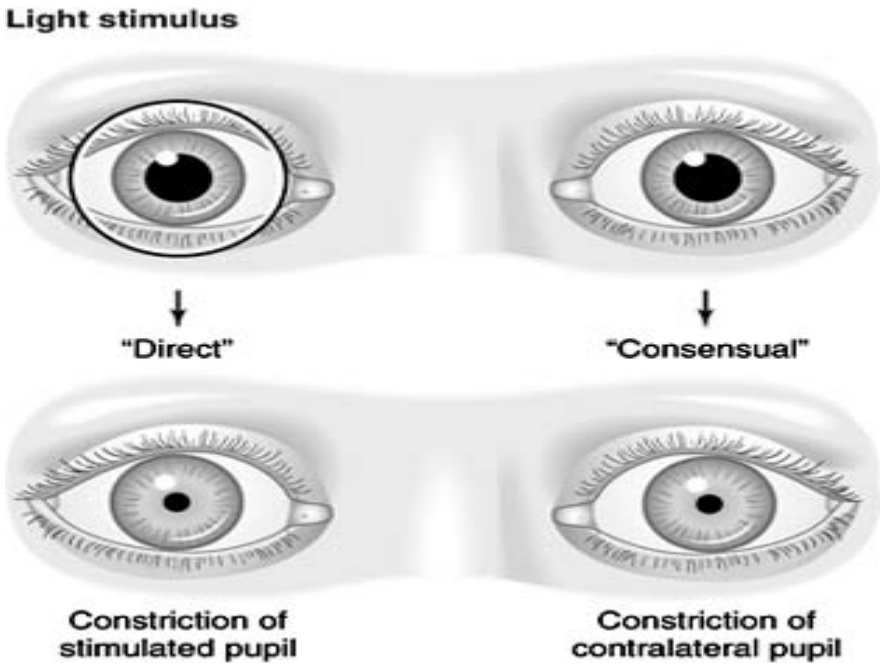
It can be variable from 6/9 to loss of perception of light. In most of the cases visual acuity is reduced . Contrast sensitivity and colour vision is defective in most of the cases.

The reduction in visual acuity parallels the reduction in contrast sensitivity.[27] RAPD is present and detectable with a swinging flash light test. Patients with optic neuritis also can be shown to have a reduced sensation of brightness in the affected eye simply by asking them to compare the brightness of a light shined in one eye and then another or by performing more complex testing with a flickering light.

2) Pupillary reflexes:

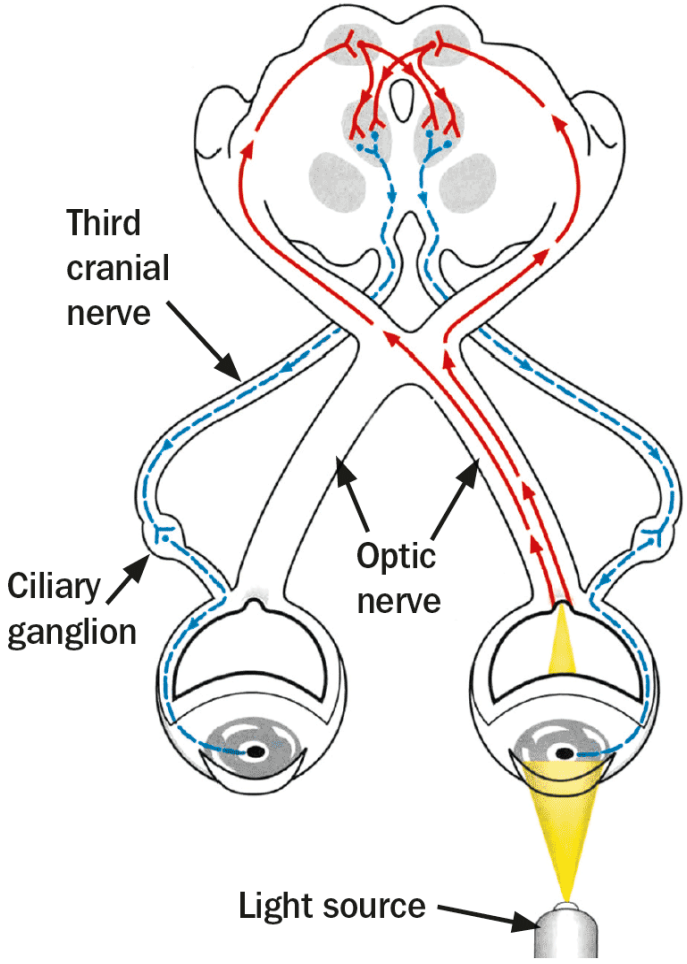
RAPD is a office room test. It determines decreased vision in patient in optic nerve problems.

Constriction of the ipsilateral pupil when the ipsilateral eye is stimulated by light, is called direct light reflex. Constriction of contralateral pupil when ipsilateral eye is stimulated by light, is called consensual light reflex.



Light reflex pathway has two pathways

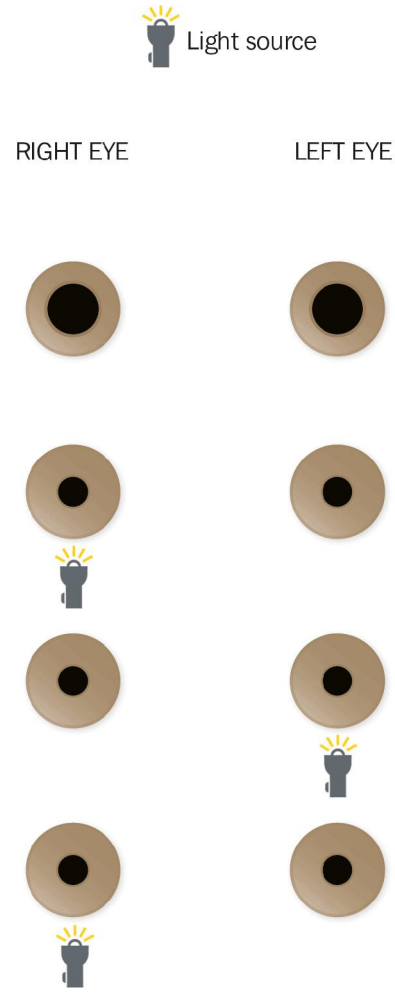
1. Afferent pathway extends from the retina along the optic nerve to the mid brain when a light is shone in that eye.(red)
2. Efferent pathway consist of parasympathetic fibres from mid brain back to both pupils via third cranial nerve and ciliary ganglion. It causes both pupils to constrict when light is stimulated.



A swinging flash light test is used to detect RAPD. It is the paradoxical response of a pupil to light.

PROCEDURE:

Patient is asked to fix at a distance. A bright flash light is shone on to one eye from below or from temporal aspect and constriction of pupil is noted that eye. Then the flashlight is quickly moved to the contralateral eye and response of the pupil is noted. This swinging to and fro is repeated several times to look for the pupil response. Normally both the pupils constrict equally and the pupil to which light is transferred remains tightly constricted. In presence of RAPD in one eye, the affected pupil will dilate (paradoxical response) when the flash light is moved from the normal eye to the abnormal eye. This response is called Marcus Gunn pupil.



Neutral density filters may be used to quantify the asymmetry in afferent input from each eye. It is used over normal eye while performing swinging flash light test. Filter placed in front of normal eye neurtralises the defect in abnormal eye. It is measured in log units.[28]

3) Colour vision

Colour vision is the ability of the eye to distinguish between colours excited by light of different wavelength. Dyschromatopsia can be congenital or acquired. Acquired loss of colour vision can be due to many causes. 88% of colour vision abnormalities was seen in optic neuritis. Mixed defects like red- green and blue- yellow were reported in ONTT trial. Blue yellow defects were more common in acute phase of disease. Red-Green defects were more seen at 6 months.

Colour is purely a subjective sensation.[29] It is mainly dependent on the wavelength composition of light entering the eye and on the structure of eye.[31]

There are three theories of colour vision

1. Opponent theory
2. Trichromatic theory
3. Zone theory

COLOUR VISION TESTS:

The main objective is to [31]

1. Screens the presence or absence of congenital or acquire colour deficiency
2. Diagnose the type and severity of colour deficiency.
3. Asses its significance in a particular vocation, employement or vocation.
4. They are designed to perform various functions
    a) Screening test- identifies subjects with normal and abnormal colour vision.
    b) Grading test- estimates severity of colour vision
    c) Vocational tests- identifies colour matching ability,hue discrimination and colour recognition.

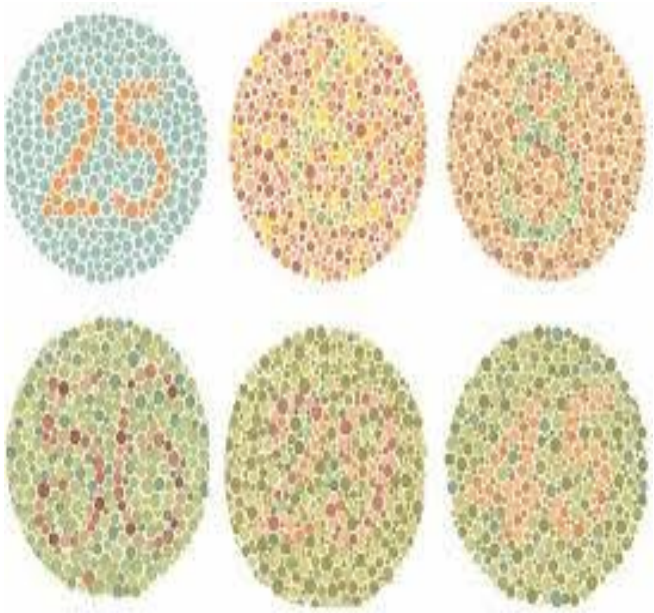
PSEUDOISOCHROMATIC PLATE

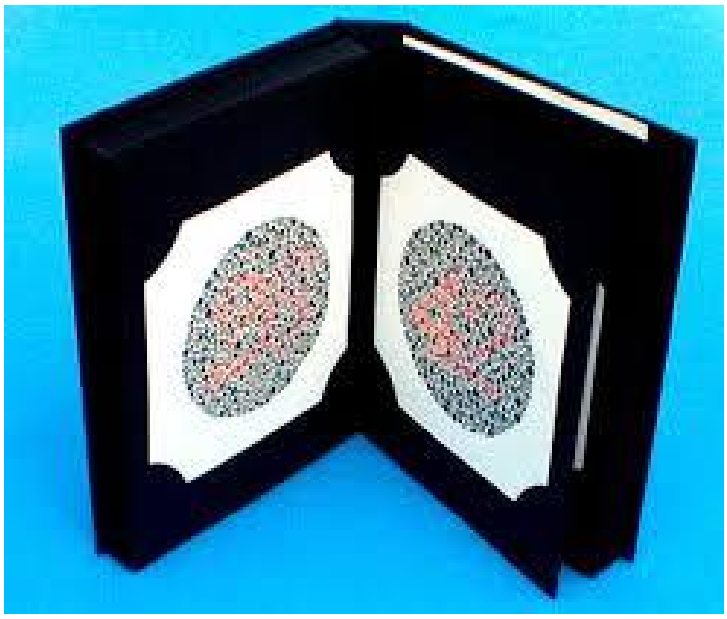
1. Ishihara plates
2. American Optical Hardy- Rand –Rittler Plates
3. Standard Pseudoisochromatic Plates
4. Colour Vision Testing Made Easy
5. City University Test.

Ishihara charts are based on the principle of confusion of the pigment colour in red –green colour defectives. It is easy and rapid test most commonly used for screening purposes. Charts are available in 14,24 or 38 plates.

INTERPRETATION:

The plates are designed to be appreciated correctly in a room which is lit adequately by daylight. The plates are held at adistance75 cm perpendicular to the line of sight. Out of 21 plates, if 17 or more plates are read correctly by an individual his colour sense is regarded as normal. If 13 or less plates are read the the person has red- green colour defect.[32]





American Optical Hardy- Rand –Ritter (HRR) is the test for choice for quantitative diagnosis.

Standard Pseudoisochromatic Plates Part 2 for acquired colour deficiency.[30,33]

It is an alternative method of colour vision assessment. It uses high resolution colour monitor. It is expensive. Designs are similar to Ishihara, HRR AND City university.

City University Colour Assesment

It is based on spatiotemporal luminance masking technique. In this, part of uniform background it is formed by spatially discrete elements that are equal in time averaged luminance with respect to the background, ensuring detection of the stimulus is based purely on chromatic discrimination and not luminance difference. This is designed to run on any monitor balanced approximately 9000K, which is the default factory setting for most colour monitors.

GRADING COLOUR VISION DEFICIENCIES

Spectral anomaloscope

1. Nagel anomaloscope
2. Oculus HMC
3. Neitz anomaloscope
4. Pickford – Nicolson anomaloscope.

Anomaloscopes are used for screening, diagnosis, classification and grading of colour vision deficiencies. It is expensive and difficult to administer. [33,38]

Arrangement tests (Hue Discrimination Tests)

1. Farnsworth Muller 100 Hue test
2. Farnsworth Muller dichotomous D 15 or Panel D 15 test
3. Lanthony desaturated D 15
4. Adams desturated D 15 test

Farnsworth Muller 100 Hue test

It is a very sensitive reliable and effective method of determining colour vision defect.

Farnsworth Muller dichotomous D 15

It is to distinguish observers with moderate or severe colour defects to those with milder defects.[30]

Lantern test

Its a colour naming test. Edridge –Green Lantern is designed to produce a range of colours and tints. It is an instrument used for testing the ability of a person to recognize colour of transmitted light. It was built to stimulate the light of railway signals as they are visible from distance.

Test is performed in a dark room at six meters distance. Set of filters showing signal red, yellow, green and blue colours.

Edridge –Green Lantern

Colour vision is almost affected in patients with optic neuritis. It is severely affected than visual acuity. Testing of colour vision is helpful in diagnosing ON in patients with minimal loss of vision. Contrast sensitivity measures the degree of detail perception in combination with perception of contrast.[29] Ishihara pseudoisochromatic color plates can detect colour vision defects in eyes with retrobulbar neuritis and can detect evidence of optic nerve dysfunction.

Marta Owidzka et al [60] reported in 27 patients of 33 eyes as contrast sensitivity is significantly reduced in all spatial frequencies both in photopic and mesopic conditions.

Contrast sensitivity:

It is the ability to perceive slight changes in luminance between regions which are not separated by definite borders. Ability to perceive sharp outlines of relatively small objects. Loss of contrast sensitivity is more important and disturbing to the patient than the loss of visual acuity. Even in presence of normal visual acuity, contrast sensitivity can be impaired.

Measurement of contrast sensitivity of human visual system was reported by Schade in form of Modulation transfer function.[34]

Campbell and Green in 1968 first measured contrast sensitivity using sinusoidal gratings and concluded that measurement of contrast sensitivity gives more complete description of function of retina.[35]

TYPES:

1. Spatial contrast sensitivity
2. Temporal contrast sensitivity

Measurement:

It is measured as (Lmax – Lmin) / (Lmax + Lmin).

L - Luminance recorded by photocells scanning across the gratings.

3 variables in measurement of contrast sensitivity:

1. Amount of light reflected depends on illumination of paper and darkness of ink.
2. Degree of blackness in relation to the white background . i.e contrast
3. The distance between the grating periods or cycles per degree of visual angle.

Bodie Wollner introduced contrast sensitivity suggesting the name ‘visuogram’ to an ‘audiogram’ to describe a patients ‘contrast sensitivity curve’.

Deficits expressed in terms of decibels. Three types of deficits were described.

1. High frequency type characterised by increasing loss at high frequency.
2. A level loss type characterised by a similar loss for all spatial frequencies.
3. A selective loss type characterised by deficits in a narrow band of spatial frequencies.

Methods to measure contrast sensitivity include simple plate [36], cathode ray tube display on a screen letter acuity charts.[46] Visual field testing using low contrast rings on stimuli, pattern discrimination test, two alternative forced choice test.

RELIABLE METHODS OF MEASURING CONTRAST SENSITIVITY

1. Arden grating
2. Cambridge low contrast gratings
3. Pelli Robson contrast sensitivity chart

Arden gratings:

Arden in 1978[36] introduced a booklet containing seven plates – one screening plate(no 1) and six diagnostic plates (no 2 -7). The contrast changes from top to bottom and covers a range of approximately 1.76 log units. Plates are studied at 57 cm, with spatial frequency increasing from 0.2 cycles /degree to 6.4 cycles/degree each being double the frequency .

Cambridge low contrast gratings:

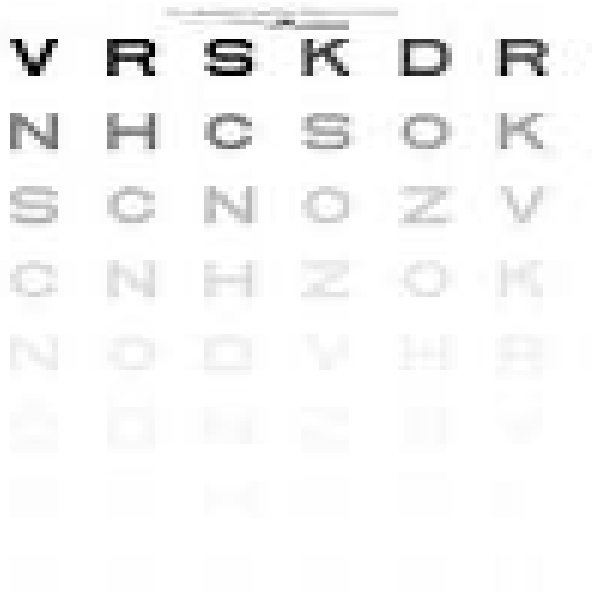
It consist of set of ten plates containing gratings in a spiral bound booklet.

To perform this, booklet is hung on wall at a 6 metres distance. The pages are presented in pairs one above the other. One page in each pair contains gratings and the other is blank. Subject is require to choose which page top or bottom contains gratings. Pages are shown in descending contrast

Pelli – Robson contrast sensitivity test:

It consists of letters which subtends an angle of three degrees at a distance of one meter. The chart is printed on both the sides. The two sides have a different letter sequence. The letters on chart are organized as triplets, there being a two triplets in each line. The contrast decreases

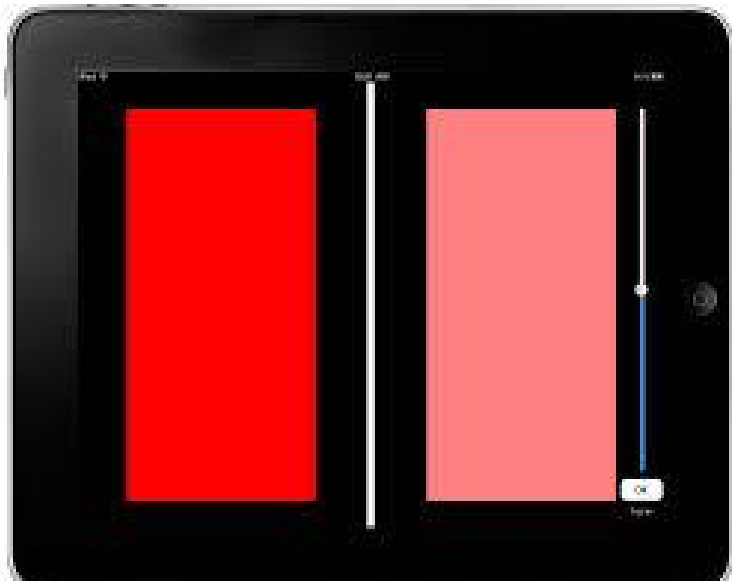
from one triplet to next. The log sensitivity varies from 0.00 -2.25. Chart is hung on the wall, it should be at the level of subjects eye. Chart is illuminated uniformly. Luminance of white area is between the accepatable range of 60 to 120 cd/m. It is recorded at 1 metre distance. Sensitivity is indicated by finest triplet for which two of the three letters are named correctly.



RED DESATURATION:

Most commonly used. It can be tested using bright coloured objects to compare the function of two eyes. Patient is asked to compare the saturation or redness of the object and indicate whether there is any less

saturated redness in either eye indicating an acquired dyschromatopsia in that eye. Its a sign of optic nerve dysfunction.



BRIGHTNESS COMPARISON:

It can be subjective or objective.

Subjective:

A bright beam of light is shone into the better eye and patient is asked to grade it as 100%. Then it is shone into the involved eye and the patient is asked to assign a percentage value to the brightness perceived in this eye as compared to normal eye. Eg 50% of the normal.

Quantification:

It is done by putting a sequence of neutral density filters in the better eye and shining the light until the patient states that the brightness is same in the both the eyes.

In 89% of patients with optic neuritis poor brightness perception was reported .[45,37.]

Visual fields:

Visual pathway extends from retina to occipital cortex. It is an extra cerebral course. Damage to the nerve fibres along the visual pathway cause variety of field defects. Complex course of nerve fibres within the visual pathway and any damage to the fibres at certain location will produce visual field defects.

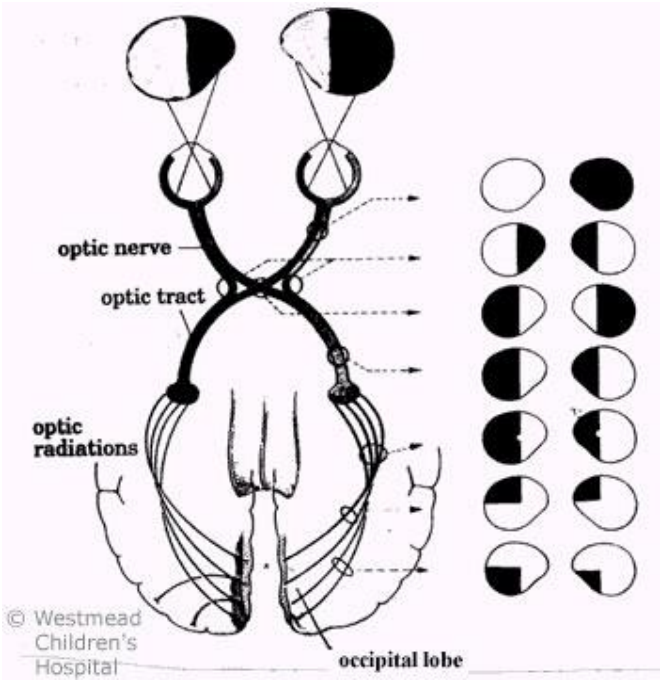


FIGURE : 1.1 Lesions along the optic pathways showing visual field defects.

Testing of central and peripheral visual fields is important:

1. To detect abnormality in vision.
2. To localize the defect along the afferent visual pathway.
3. To quantitate the defect and measure the change over time.

Visual field testing can be divided into static and kinetic which can be manual or automated. Automated perimetry lacks field defects shape details, but it is proved to be more sensitive than kinetic testing.

The following are the methods in visual field testing

a) Confrontation methods
b) Amsler grid
c) Tangent screen
d) Goldmann perimetry.

Confrontation methods:

This is a rapid screening method where examiner compares patients field with their own.

Finger counting:

Patients are instructed to accurately identify the number of fingers presented in each quadrant of the monocular field.

Simultaneous finger counting may bring out a subtle defect.

Test :

Patient covers one eye with the palm of one hand and look at the examiners nose a feet away. The examiner closes his eyes opposite from the patients covered eye and have the patient fixate on the examiners open eye.

The patient while fixating should be asked whether he can see the examiners eyes, ears, hair, mouth etc which would reveal a large scotoma.

Hand comparison :

Simultaneous perception of hands placed on either side of vertical meridian providing a sensitive subjective comparison of the two hemifields.

Hands placed in the superior and inferior fields to determine whether there is an altitudinal defect.

Colour comparison :

Done in similar way with the patient occluding one eye. The examiner holds a red coloured object in each hand and asks whether the red objects appear the same in each hand. In a relative scotoma one object might appear less red than the other.

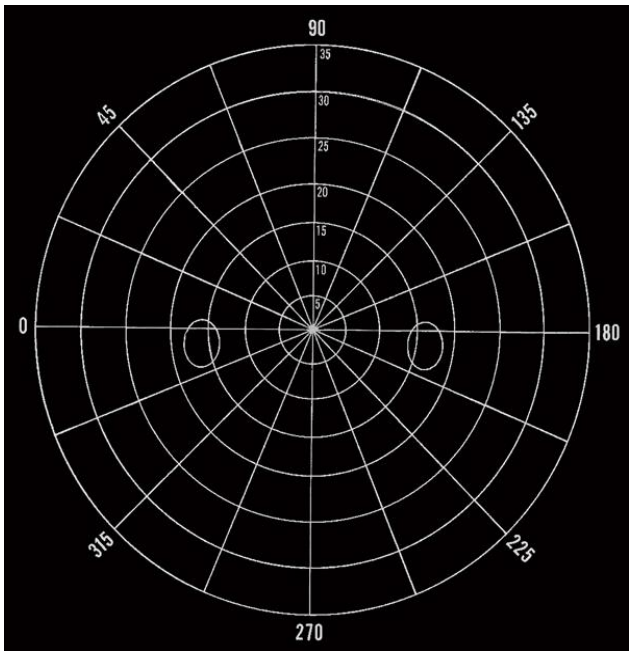
A central scotoma of the field can be detected by asking the patient to describe changes in the saturation of the colour of a large object moved away or toward central fixation.

b) Amsler grid :

Screens central 10 degree of fixation. Patient optically corrected for near vision covers one eye and looks at fixation point in the centre of a grid. Patient is then asked to describe any scotoma.

c) Tangent screen:

It is a valuable sensitive method of evaluating or screening visual field defects. It offers relative magnification of the surface area at 1 or 2 m (when compared to other perimeters at5 33 cm). It allows detailed exploration of small central scotomas. Tangent screen is less quantitative and standardized.



Method:

The patient gazes with one eye at black screen with the other eye occluded 1- 2 metres away in good lighting and fixes on a central spot. Thin black wand with various targets on tip is used by the examiner. Initially a suprathreshold stimulus is used such as 5mm white target at 1m. stimulus is moved from non seeing area to seeing area.

The target is a flat disc white or red on one side, black on reverse so that it can be flipped over with course of the test so that black surface is not seen by the patients it blends with the screen. Patients are instructed to indicate verbally or by gesture when they first see the target only, not the wand or examiners hand.

Blind spot can be detected using larger stimulus. Central fields(fixation area) explores scotoma in the central region of fixation and the area between blind spot.

d) Goldmann perimetry:

It is useful for evaluating both central and peripheral fields. The perimetry can be used along with a moving target (kinetic) or stationary target (static perimetry). It is standardized and reproducible target and background luminance (i.e contrast). Humphrey Visual Field Analyser

(HVFA) sensitivity mainly requires patient's co-operation and competence, good technician to monitor the patient.

In ONTT, patients underwent HVF testing. Perimetry revealed that 48% present with a diffuse field defect (Figure 1.2), 20% with altitudinal or arcuate defects and 8% present with a centrocaecal defect [44.] . Initially it was found that centrocaecal scotoma was common defect in optic neuritis. However it is now believed that the central scotoma found by Goldmann perimetry represents diffuse suppression of sensitivity within the central 30 degrees of vision.[43] Central visual loss is common in optic neuritis patients and sparing of the periphery, it is rare for the peripheral field to be abnormal in the presence of a normal central field.

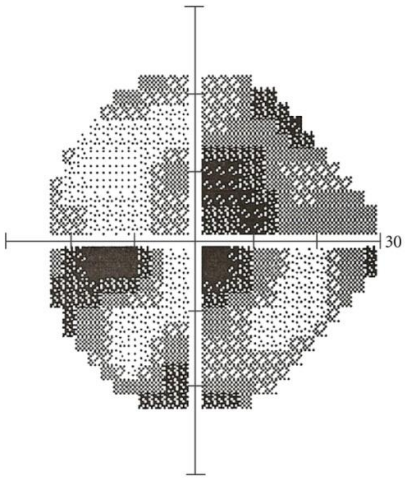


Figure 1.2: Humphrey visual field analysis showing diffuse loss of sensitivity with a dense central scotoma in optic neuritis.

Other test are

1) Stereoacuity :

The Titmus Polaroid 3D Vectograph stereoacuity is recommended for both children and adults with optic neuritis.

The Pulfrich effect in which patient experience a stereoillusion by having the patient gaze at a pendulum swinging at right angles to the line of sight and determining if the pendulum appears to the patient to be swinging in an elliptical path is a sensitive indicator.

2) Visual Evoked Potential:

It a specific change in Electroencephalographic (EEG) recording due to stimulation of the visual pathway to either a pattern or flash stimulus. Recordings of evoked potentials can be made with the use of electrodes applied to the scalp. The main signals are detected over the occipital cortex. The VEP signal is mainly derived from the macular region as the predominant function of the occipital cortex is to subserve macular function.

It differentiates organic from functional cause of defective vision. It tests central and perifoveal visual field and there is prolongation of P latency which is a permanent change. The P100 amplitude is correlated

with visual acuity and P latency is also an indicator of optic nerve dysfunction. In pattern shift VEP it provides evidence of optic nerve pathology in optic neuritis.

3)Pattern electroretinogram(PERG)

It monitors integrity of central ganglion cell layer. It is of value in improved interpretation of abnormal VEP pattern when both are recorded simultaneously to rule out if delay in pattern VEP P latency in a patient with suspected optic nerve demyelination.

4)Pupillary light reflex latency:

Prolonged latency of papillary light reflex which is measured using infrared reflection.

5)Foveal critical flicker frequency is impaired

Subjective brightness measured by Authorn Flicker test in relation to flicker frequency is abnormal.

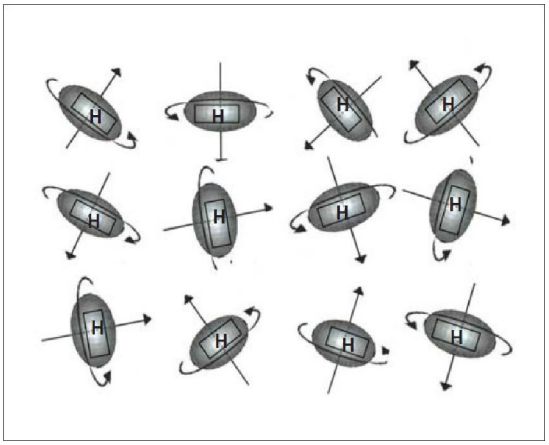
OPTIC DISC FINDINGS:

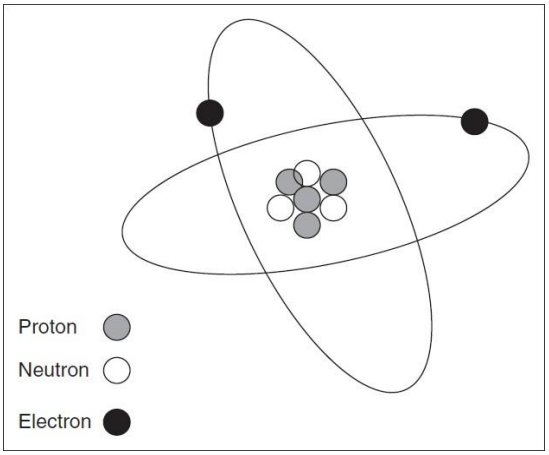
- Normal in retrobulbar neuritis
- Swollen disc in papillitis.
- Marshal et al reported Central or paracentral scotoma with or without peripheral extension represents 90% of field defects.

MAGNETIC RESONANCE IMAGING:

PRINCIPLE:

MRI works on basis of hydrogen atom which is the important atom present in the body. MRI imaging is mainly based on the movement of hydrogen protons in magnetic field. Protons are stimulated by the radiofrequency coil and then left to relax in the direction of magnetic field creating energy that is used for development of images.





MRI shows the size ,quantity and distribution of lesions larger than 2mm, and together with supporting evidence helps in the diagnosis of MS.

Magnetic Resonance Imaging is helpful in diagnosis of clinically silent multiple sclerosis lesions.

MRI criteria for diagnosing MS

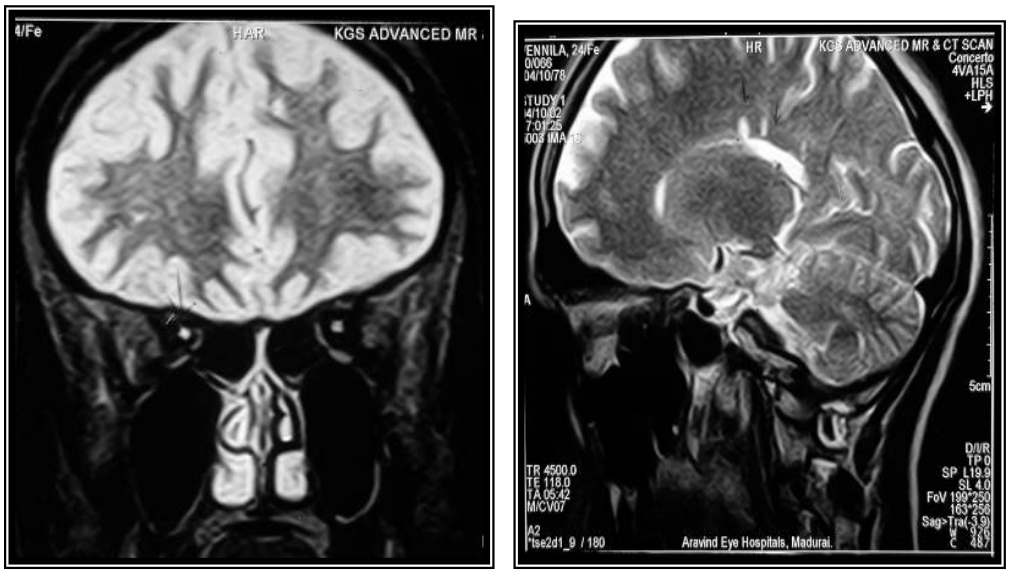
At least 3 lesions and two of the following should be present for the diagnosis of MS to be present;[39]

1. 1.Lesions in the lateral ventricles
2. Lesions with diameters greater than 5mm
3. Lesions present in posterior fossa (infratentorial).

MRI features in optic neuritis:

MRI shows typical periventricular and corpus callosum plaques in multiple sclerosis. The plaques typically have an ovoid shape with their long axis perpendicular to the ventricular margins. Acute demyelinating lesions may be highlighted with gadolinium on T1 weighted scans.

40 – 70 % of patients are reported to have periventricular white matter changes on MRI consistent with MS in cases of isolated optic neuritis. In ONTT, MRI scans were the strong predictor of developing MS performed at study entry. 10 year risk of MS ranging from 22% in patients with no MRI lesions, 56% in patients with one or more lesions.[41]



Optical Coherence Tomography:

It is a non invasive measurement using infrared light to reflect retinal thickness which is associated with axonal and neuronal degeneration. [40,42].

SYSTEMIC INVESTIGATIONS

1. Routine hemogram
2. X-ray (Chest)
3. Mantoux
4. FTA - ABS & VDRL for syphilis
5. serology and culture for bartonella
6. Markers of viral infection
7. Serum electrolytes and fasting blood sugar
8. MRI
9. Lumbar puncture and CSF tap (IgG index and oligoclonal bands)
10. Blood culture
11. ANA, dsDNA
12. Serology for toxoplasmosis

DIFFERENTIAL DIAGNOSIS

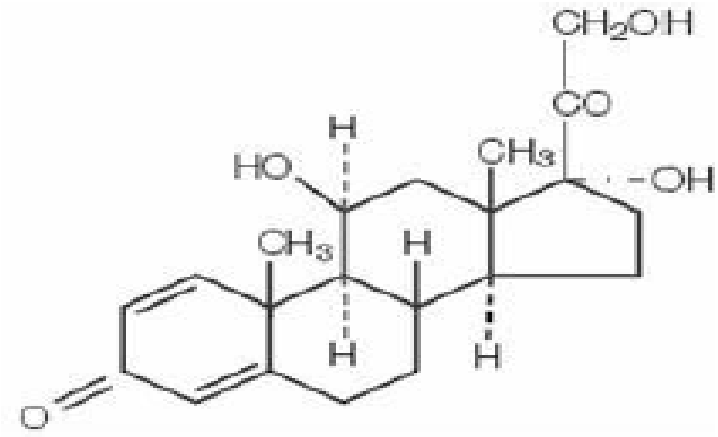
1. Ischemic optic neuropathy, it is characterized by lack of pain, pallid disc swelling with haemorrhages. Altitudinal visual field defect. Lack of improvement of vision with standard therapy.
2. Lebers optic neuropathy it is characterized by lack of pain, circumpapillary telengiactatic microangiopathy
3. AION
4. Toxic or nutritional deficiency amblyopia
5. Non organic visual loss
6. Other
    a. posterior uveitis
    b. CSCR
    c. Disc drusen
    d. Glaucoma
    e. Posterior scleritis

MANAGEMENT

Corticosteroids are main stay of treatment. It can be oral, parenteral or retrobulbar route.

PREDNISOLONE:

It is a adrenocortical steroid which are naturally occurring and synthetic,readily absorbed from the gastrointestinal tract. It is a white crystalline powder very slightly soluble in water. It is designated chemically as pregna-1,4-diene-3,20-dione,11,17,21-trihydroxy-(11 β). The structural formula is represented below:



Mechanism of action:

It suppress inflammatory and immune response and alter leucocyte function.

METHYLPREDNISOLONE:

It is steroid that prevents the release of substances in the body that cause inflammation.

COMPLICATIONS OF STEROIDS:

OCULAR:

Eyelids:

a) allergic reactions

b) telengiectasia

c) persistent erythema

Cornea:

a) Superficial punctuate keratitis

b) Delayed healing of corneal wounds

GENERAL

1. Increased IOP
2. Delayed healing of corneal wounds.
3. Mydriasis – precipitate angle closure glaucoma

4. complicated cataract
5. Enhances lytic action of collagenase

SYSTEMIC COMPLICATIONS:

A) Dermatological :

1) Acne
2) Hirsuitism
3) Subcutaneous tissue atrophy

B) Immunological:

1) Impaired inflammatory response
2) Delayed tissue healing

C) Metabolic:

1) Secondary diabetis mellitus
2) Hyperosmotic ketoacidosis
3) Centripetal obesity
4) Hyperlipidemia

D) Cardiovascular:

1) hypertension
2) sodim and fluid retention

E) Musculoskeletal:

1) osteoporosis
2) vertebral compression fracture

F) Gastrointestinal:

1) peptic ulcer
2) gastric haemorrhage
3) intestinal perforation
4) pancreatitis

G) Endocrine:

1) Adrenal insufficiency
2) Cushing s syndrome
3) Growth failure
4) Menstrual disorders

H) Neuro Psychiatric:

1) Pseudotumour cerebi
2) insomnia
3) mood swings
4) psychosis

OPTIC NEURITIS TREATMENT TRIAL:

ONTT is a multicentered randomized trial involving 454 patients from 1988 to 2006. This study evaluated the efficacy of corticosteroid treatment for acute optic neuritis.

Study objective:

It evaluates the efficacy of corticosteroid treatment in acute optic neuritis relationship between optic neuritis and multiple sclerosis.

The patients in this study were randomized into 3 different groups

1. IV methyl prednisolone(250 mg 6 hrly) for three days followed by oral prednisolone for 11 days and three day tapering of prednisolone.
2. Oral prednisolone (1mg/kg/day) for 14 days followed by 3 days in tapering doses.
3. Oral placebo for 14 days.

Results:

Intravenous methyl prednisolone 1gram followed by Oral Prednisolone 1mg/kg speeds the recovery of visual loss due to optic neuritis and results in a slightly better vision at six months.

STUDIES INVOLVED IN OPTIC NEURITIS TREATMENT:

CHAMPS STUDY (The Controlled High Risk Avonex Multiple Sclerosis Trial)

Objective :

Avonex (interferon beta 1 a) treatment would benefit patients who had experienced a demyelinating event involving the optic nerve, brain stem/cerebellum or spinal cord. Previously,brain abnormalities in MRI predicted likelihood of future MS like events. All patients received intravenous methylprednisolone 1g per day for three days within the days of onset of neurological symptoms, followed by oral prednisolone with tapering dose of 1mg/kg for 11 days. Patients were divided into two groups

Group 1 once weekly intramuscular injection of interferon beta 1 A

Group 2 placebo injections

Outcome was measured by development of CDMS and change in demyelinating lesions on serial brain MRI scans.

Results

At three years end,the probability of CDMS was 50% in the placebo treated group and 35% in the interferon 1 A treated group. There was no difference among treatment options among patients presenting with optic neuritis, brain stem/cerebellar or spinal cord events. Treatment with Avonex significantly reduces the two year likelihood of future neurological events and worsening of brain MRI in patients with a first acute CNS demyelinating event.

ETOMS (Early Treatment Of Multiple Sclerosis Study)

Objective:

To determine whether an early treatment with interferonb 1 b is effective in delaying the development of CDMS after the first attack.

Study entered 308 first attack patients, ages 18-40 years with unifocal or multifocal (39%) CNS presentations and abnormal brain MRI. Patients were assigned to receive IFN beta 1 A 22mcg SC weekly or placebo. These patients were followed for 2 years.

CHAMPIONS STUDY (Controlled High Risk Avonex Multiple sclerosis Prevention Surveillance)

Objective:

Outcomes are compared in those who had given drug from the start of CHAMPS study (immediate treatment) versus those who had switched from placebo after about 30 months. (delayed treatment).

Results:

Immediate group had few relapses and fewer MRI lesions than the delayed group and few of its members converted to definite MS.

BENEFIT STUDY: (Betaferon in Newly Emerging MS for Initial Treatment)

Beta interferon 1b double blind , placebo controlled trial in patients with first episode suggestive of MS. Results showed risk of developing MS within a year was reduced by 46% .

Newer modalities:

It can modify the disease course

1. Copolymer 1
2. T cell receptor peptide immunisation
3. Anti CD 4 monoclonal antibody
4. Azathioprine (Imuran)
5. Cyclophosphamide (cytoxan)
6. Oral myelin
7. Methotrexate
8. Cladribine
9. Intravenous immunoglobulin G

AIM AND OBJECTIVES

To study the visual function, response to treatment and visual outcome in patients with optic neuritis presenting to the Neuroophthalmology clinic at Aravind Eye Hospital, Madurai, South India.

MATERIALS AND METHODS

- Prospective observational case study.
- To analyse visual function, response to treatment and visual outcome in patients with optic neuritis.
- Study period: January 2013 to December 2013.
- Study duration: 13 months (including followup)
- Data source: Hospital records. (Aravind Eye Hospital)
- Sample size: 88 patients /98 eyes.
- Follow up period of 2 weeks, 1 month.

INCLUSION CRITERIA:

- Age 16 -65 years
- Loss of visual acuity or visual field, with or without pain < 1month duration.
- Unilateral / bilateral
- Fundus changes
- Field defects

EXCLUSION CRITERIA

- Age < 16 years
- Visual loss including toxic, metabolic, vascular, hereditary, compressive neuropathies.
- Previous episode of optic neuritis in the affected eye.
- Informed consent was obtained from all patients.

CLINICAL EVALUATION:

A series of 98 eyes of 88 patients who presented to our Neuroophthalmology department were included in our study.

Optic neuritis was diagnosed based on history and clinical examination which included defective vision , dychromatopsia, pain on eye movements, presence of Relative Afferent papillary defect, normal or swollen optic disc on fundus examination of less than 1 month duration were included in our study. All these patients underwent a thorough ophthalmological and neurological evaluation.

Patient ‘s data such as name, age , sex, address were documented in a proforma.

Detailed history of each and every symptom like onset, duration, pain on eye movement , headache were documented. Patients were also enquired about history of prior visual loss.

Each one of the patient included in our study have to undergo

- Visual acuity by means of Snellens’s chart
- Refraction
- Pupillary reaction to look for RAPD, sluggish pupil
- General ophthalmic examination by torch light and slit lamp.

- Intraocular pressure measurement for patients above 40 years of age by Non contact tonometry method.
- Fundus examination by direct ophthalmoscope and slitlamp biomicroscopy using +90 dioptre lens.
- Colour vision was assessed in detail by Ishihara's colour vision chart, red desaturation, brightness sensitivity.
- Central fields tested by Bjerrums's tangent screen.
- Neuroimaging was done depending upon the need and affordability of the individual patients.
- Basic blood investigations like random blood sugar, total leucocyte count, differential leucocyte count and erythrocyte sedimentation rate was done for all patients.
- All patients in our study received 1 gram of intravenous methyl prednisolne for 3 days followed by oral prednisolone of 1mg/kg/body weight for 2- 4 weeks.
- Improvement in Patient's visual acuity, colour vision, brightness sensitivity, red desaturation , visual fields and fundus were assessed during follow up examinations at 15 days and 1 month.

ANALYSIS

Analysis of collected data was done based on the following

1. Incidence
2. Age and sex distribution
3. Onset of symptoms
4. Presenting visual acuity
5. No PL/ PL / HM /1/60
6. 1/60 – 6/60
7. 6/60 – 6/6
8. Pupil - normal / RAPD.
9. Colour vision - Normal / defective / inconclusive because of poor vision.
10. Central fields
11. Fundus status – normal / abnormal.
12. Visual acuity, colour vision , red desaturation, brightness sensitivity, central fields at follow up.
13. Fundus status at follow up.

STATISTICAL METHODS

Mean (SD) or Frequency (percentage) was used to describe summary data. Wilcoxon signed rank sum test was used to assess the difference between paired data. McNemar's test was used to assess the difference between pre-post binary data. P-value is less than 0.05 considered as statistically significant. All statistical analysis was done by statistical software STATA 11.1 (Texas, USA).

RESULTS

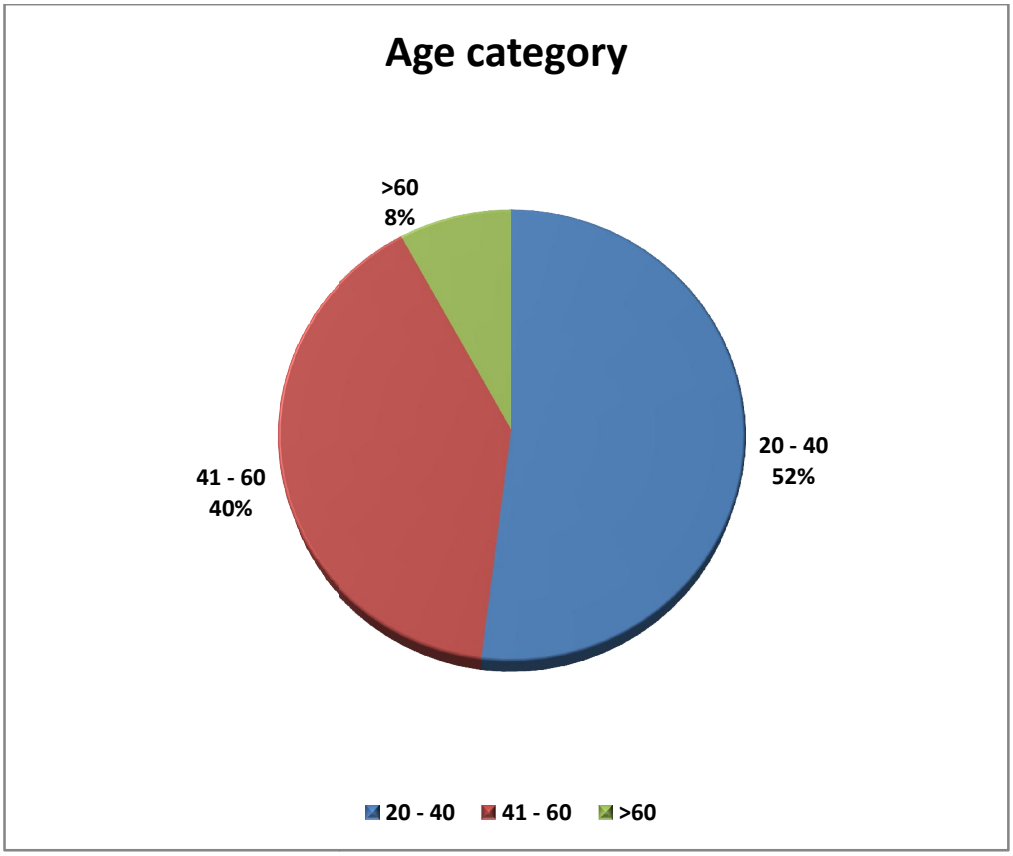
Study included 98 eyes of 88 patients with optic neuritis. Visual function parameters showed rapid recovery following treatment with steroids compared to pretreatment levels in our patients.

TABLE 1. INCIDENCE

Cases	**n**	**Incidence of Optic Neuritis**
Total number of Neuro ophthalmology cases during the study period	5,394	16.3 patients per 1000
Total number of new cases with optic neuritis	88	

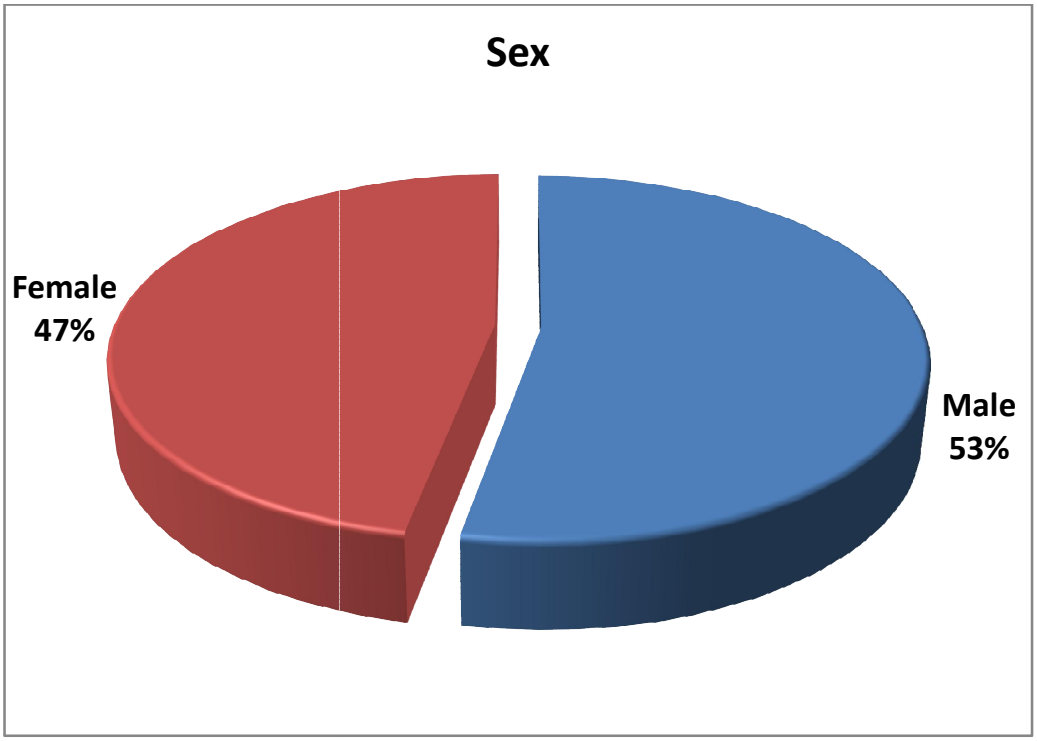
TABLE 2: AGE

The mean age is 40.0 (12.9) years and the range is 20 to 64 years.



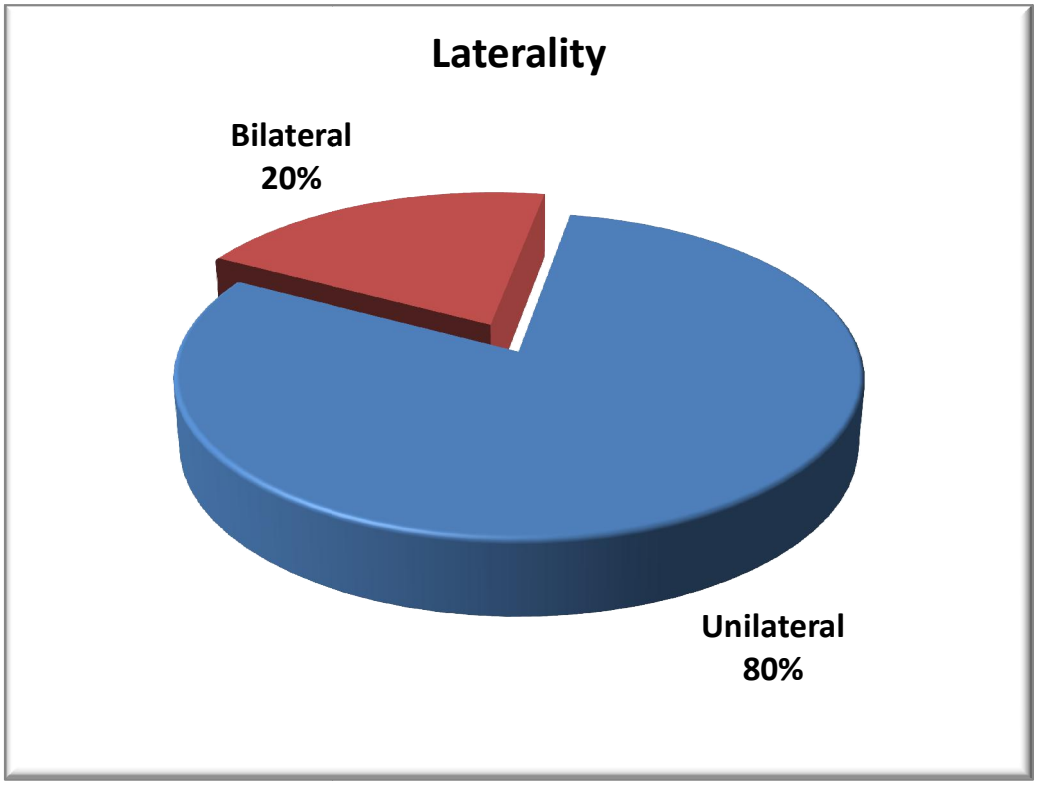
Majority of patients in the study belonged to the age group 20- 40 years. (52.3%)

TABLE 3: GENDER



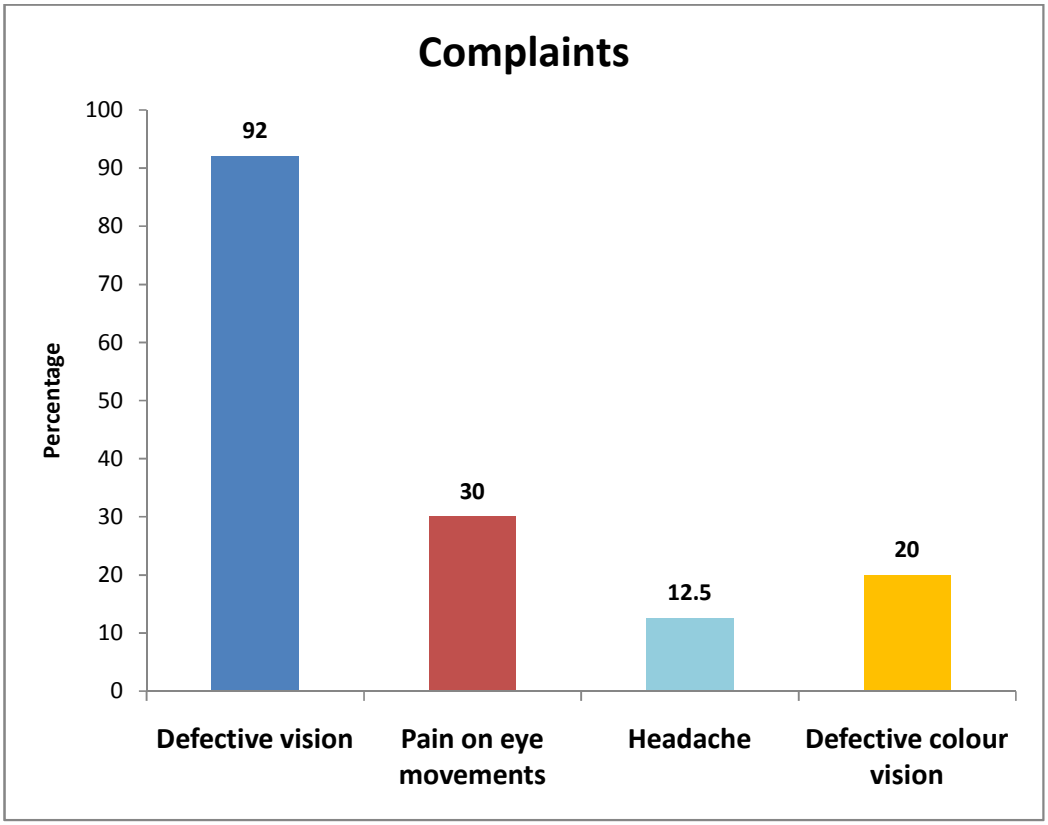
Out of 88 patients 47 were males (53.4%) and 41 were females (46.6%)

TABLE 4: LATERALITY



Out of 88 patients, 78 (79.6%) of them showed unilateral involvement and 10 (20.4%) showed bilateral involvement.

TABLE 5: COMPLAINTS

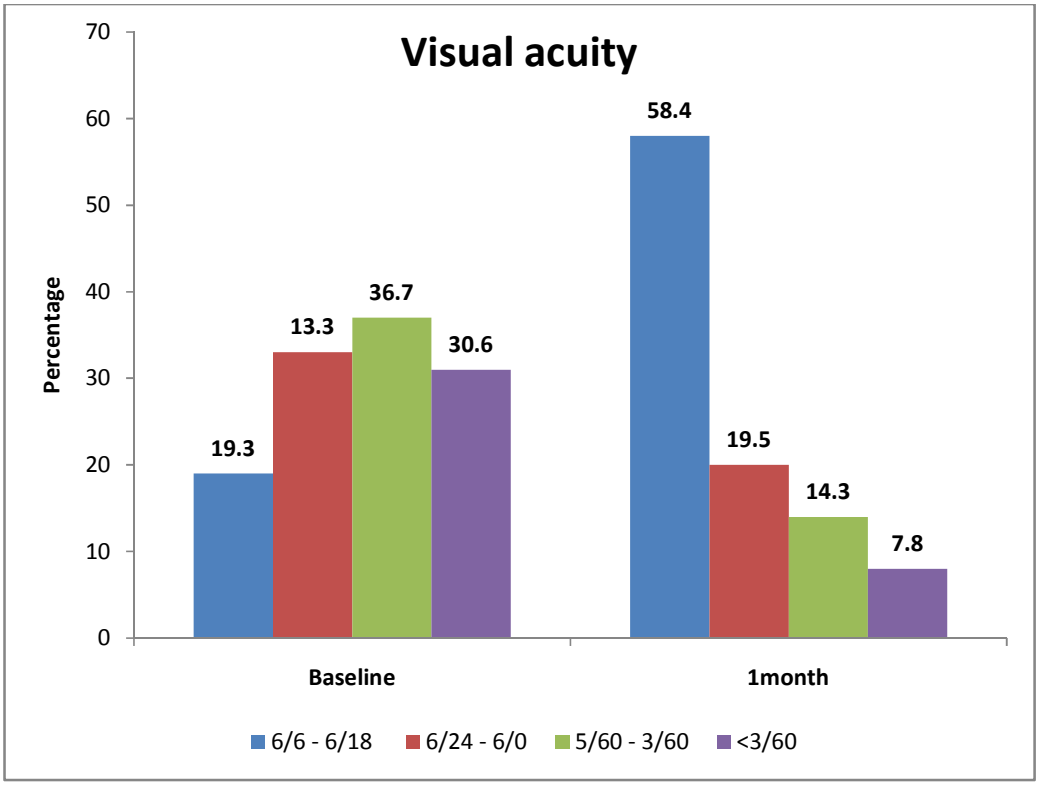


Defective vision was presented in 90 eyes (91.8%) followed by dyschromatopsia in 20 eyes(20.4%). Pain on eye movements was presented in 29 eyes (29.6%). Headache was presented in 11 patients (12.5%).

TABLE 6:VISUAL ACUITY

Visual acuity	Baseline	1month
6/6 – 6/18	19(19.4)	45(58.4)
6/24 – 6/60	13(13.3)	15(19.5)
6/60 – 3/60	36(36.7)	11(14.3)
<3/60	30(30.6)	6(7.8)
Total	98	77

At baseline 32.7% of patients had V/A >6/60. 67.3% patients had V/A <6/60. About 78 % of patients had 6/60 or better vision at 1 month follow up.



Log mar vision	N	Median (Snellen'SEquivalent)	Mean(SD)	Min-Max	P-Value
Baseline	98	1.48(2/60)	1.54(0.94)	0 – 3.2	-
2 weeks	92	0.39(6/15)	0.74(0.84)	0 – 2.9	<0.001
1 month	77	0.3(6/12)	0.67(0.83)	0 – 2.9	<0.001

Improvement in visual acuity was statistically significant at 1 month (p = <0.001) compared to the baseline.

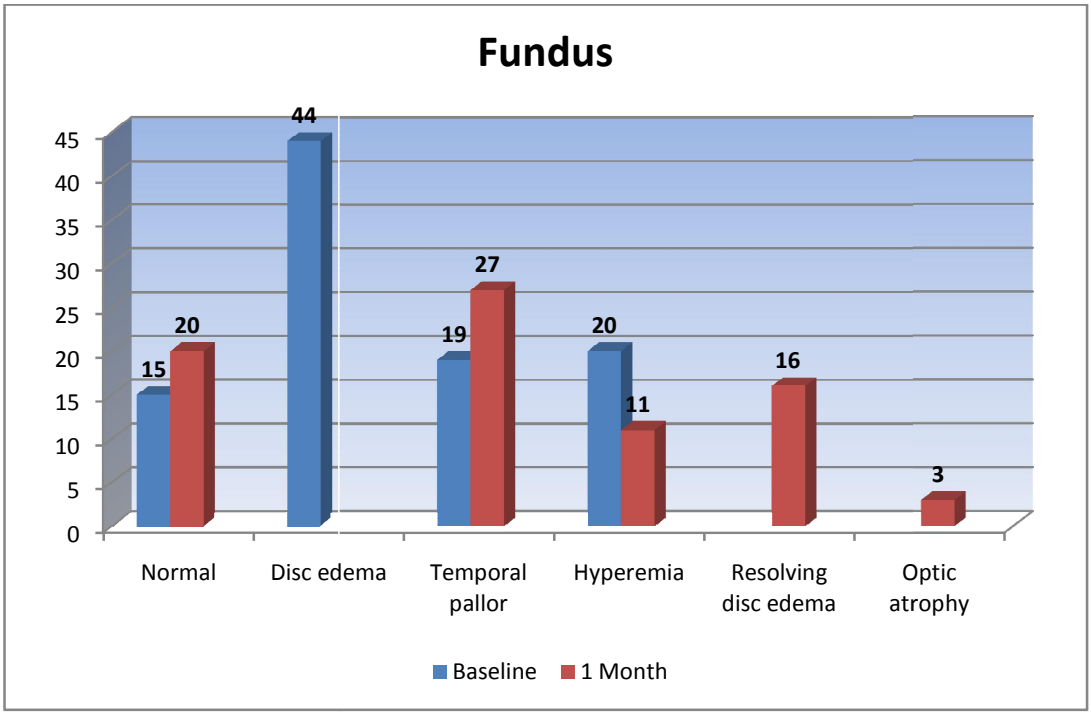
Baseline median LogMAR visual acuity was 1.48 which improved to 0.3 after 1 month.

PUPIL

Pupil	N	%
Normal	12	12.2
RAPD	86	87.8
Total	98	100.0

At baseline 12.2% showed normal pupillary reaction. 87.8% showed RAPD.

TABLE 7: FUNDUS

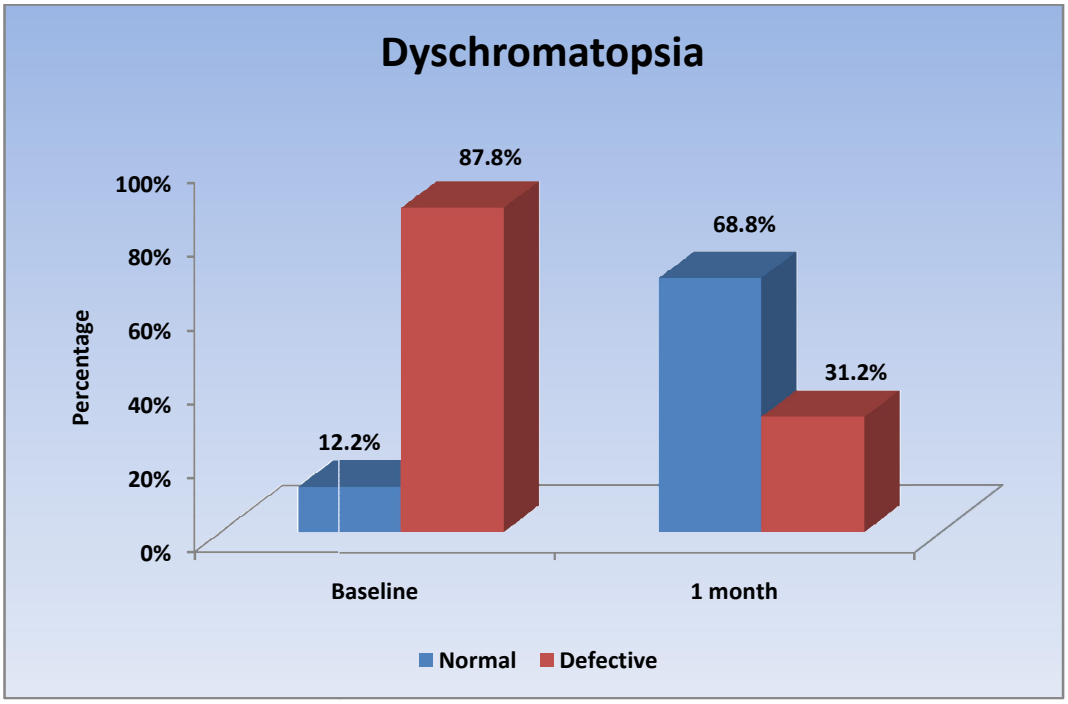


At baseline, in 15 eyes optic disc was normal, 44 eyes showed disc edema, 20 eyes showed temporal pallor, hyperemia in 20 eyes. At 1 month, out of 77 eyes 19 eyes was normal, 28 eyes showed temporal pallor, 12 eyes showed hyperemia, 16 eyes showed resolving disc edema, 3 eyes showed primary optic atrophy

Fundus at baseline	**Fundus at 1month**		**Total**	**P-value**
	Normal	Abnormal		
Normal	9(69.2)	4(30.8)	13	0.180 (Using McNemar's test)
Abnormal	10(15.6)	54(84.4)	64	
Total	19	58	77	

At baseline, 98 eyes were assessed and 15 eyes (15.3%) were normal and others were abnormal. At 1 month follow up of 77 eyes (remaining 21 were lost to follow-up), 19 eyes (24.7%) were only normal and others were abnormal. 15.6% (10) out of 64 patients were normal at final visit which were abnormal at baseline visit. At 1 month follow up, fundus did not show statistically significant improvement compared to baseline (P value = 0.180, Using McNemar's test).

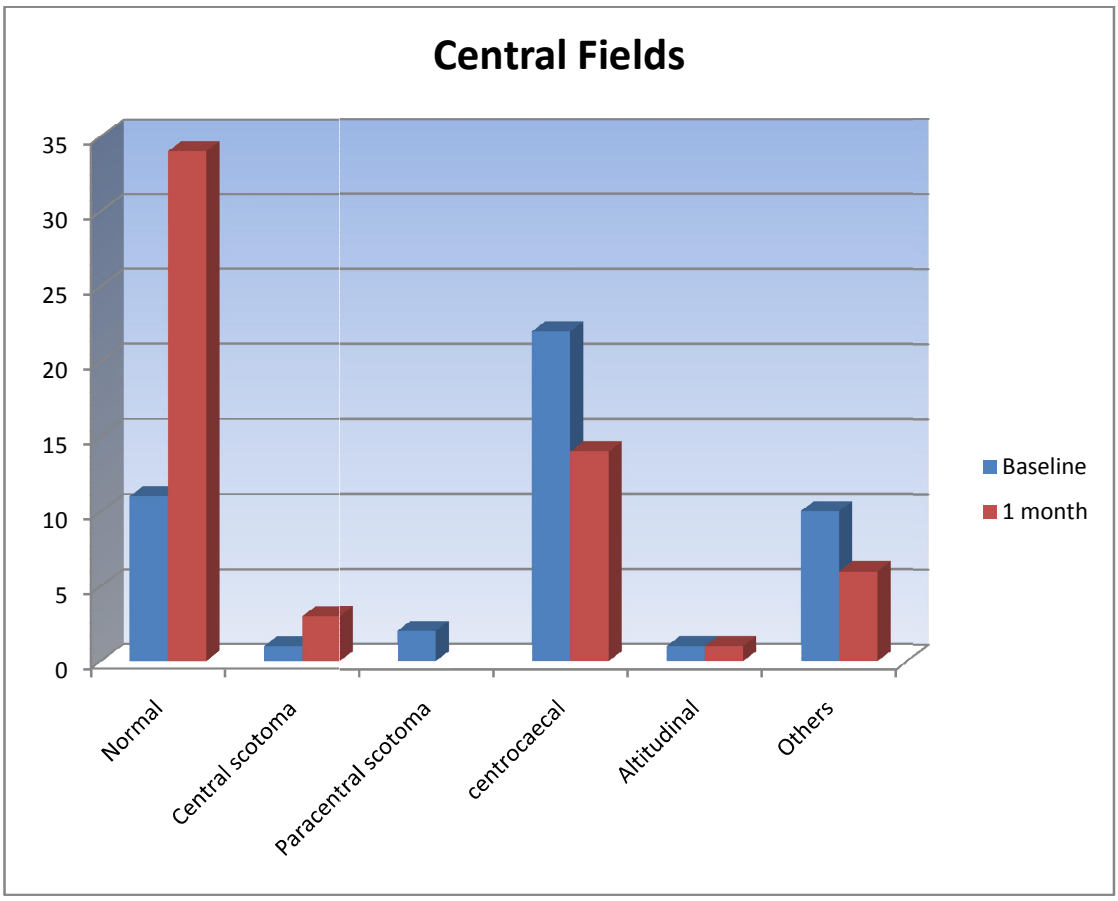
TABLE 8:DYSCHROMATOPSIA



Colour vision at baseline	Colour vision at 1month		Total	P-value
	Normal	Abnormal		
Normal	7(87.5)	1(12.5)	8	0.0035 (Using McNemar's test)
Dyschromatopsia	46(66.7)	23(33.3)	69	
Total	53(68.8)	24(31.2)	77	

At baseline 12.2% (12) were normal 87.8% (86) were abnormal. At follow up of 1 month of 77 eyes 68.8% (53) were normal and 31.2% (24) were abnormal . 46 eyes (66.7%) were normal at 1month follow-up which was abnormal in baseline. Colour vision at 1month follow-up statistically showed significant improvement. (P-value=0.004, Using McNemar's test)

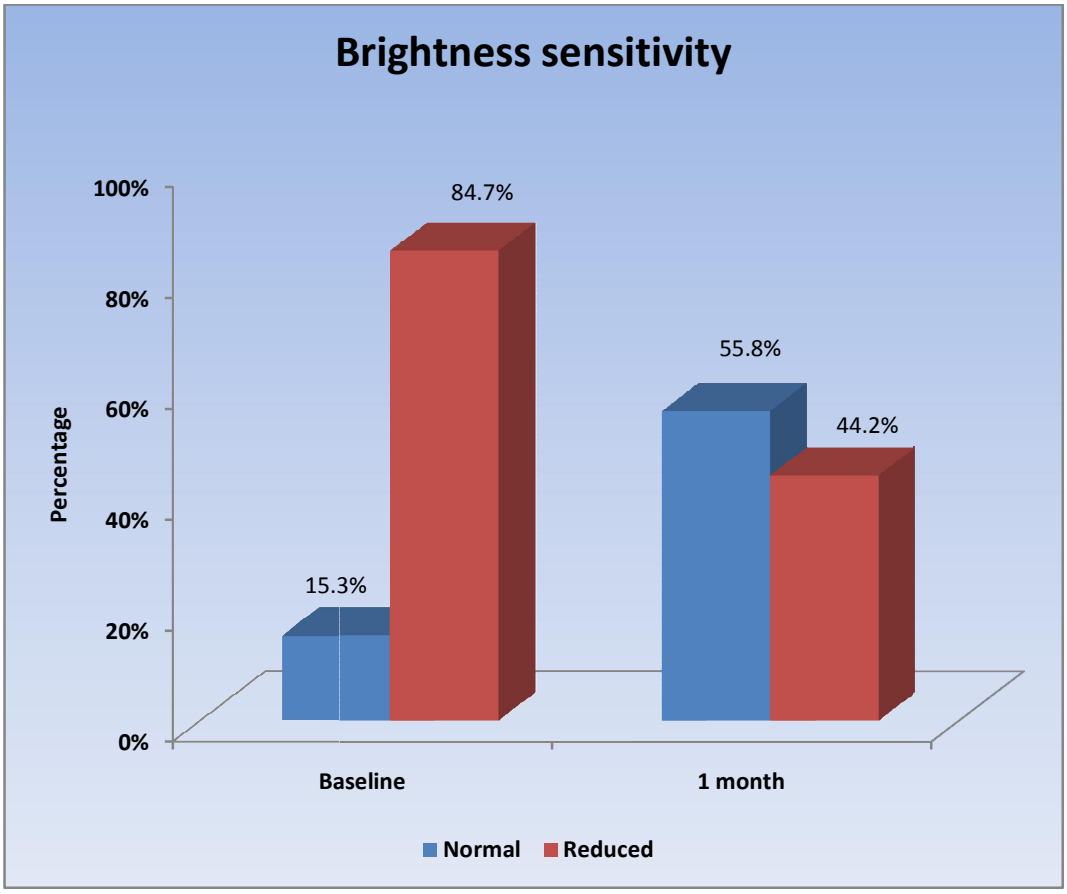
TABLE : 9 CENTRAL FIELDS



Central fields at baseline	Central fields at 1month		Total	P-value
	Normal	Abnormal		
Normal	7(100.0)	-	7	<0.001 (Using McNemar's test)
Abnormal	29(41.4)	41(58.6)	70	
Total	36(46.8)	41(53.2)	77	

At baseline 12.2% (12) were normal and at 1month 45.3% (34) were normal. 29(41.4%) eyes were normal at 1month follow-up who were abnormal at baseline. Central fields statistically showed significant improvement at 1month follow-up visit (P-value <0.001, Using McNemar's test)

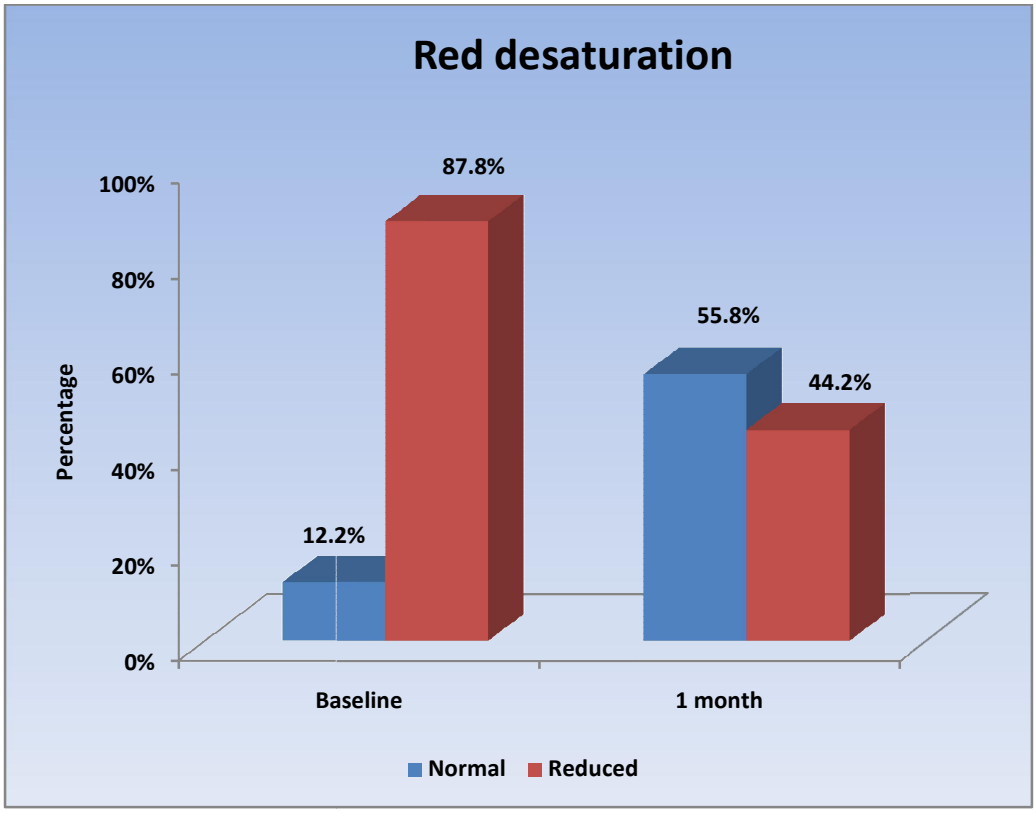
TABLE 10: BRIGHTNESS SENSITIVITY



Brightness at baseline	Brightness at 1month		Total	P-value
	Normal	Abnormal		
Normal	8(80.0)	2(20.0)	10	0.0001 (Using McNemar's test)
Reduced	35(52.2)	32(47.8)	67	
Total	43(55.8)	34(44.2)	77	

At baseline, 15.3% (15) were normal 84.7% (83) were abnormal. At follow up of 1 month out of 67 eyes 43(55.8%) eyes showed improvement. 35(52.2%) eyes were normal at 1month follow-up who were abnormal at baseline. Brightness sensitivity statistically showed significant improvement at 1month (P-value=0.0001, UsingMcNemar's test).

TABLE 11: RED DESATURATION



Red Desaturation at baseline	Red Desaturation at 1month		Total	P-value
	Normal	Abnormal		
Normal	6(75.0)	2(25.0)	8	0.0001 (Using McNemar's test)
Reduced	37(53.6)	32(46.4)	69	
Total	43	34	77	

At baseline, 12(12.2%) eyes were normal. At follow up of 1 month, 43 (55.8%) eyes showed improvement. 37(53.6%) were normal at 1month follow-up who were abnormal at baseline. Redsaturation statistically showed significant improvement at 1month follow-up (P-value=0.0001, Using McNemar's test).

Neuroimaging

Neuroimaging was not possible in all cases due to financial constraints. It was performed in 35 cases. MRI was done in 23 cases and CT was done in 12 cases. No lesions was seen in 7 cases, 25 had shown thickening and enhancement of optic nerve in the affected eye. Demyelinating lesions in the brain were present in 3 cases.

DISCUSSION

Optic neuritis is inflammation of optic nerve which is mostly idiopathic in nature.

In natural course of optic neuritis spontaneous recovery occurs within 1 week but it may take longer time in few cases. Typical cases of optic neuritis due to multiple sclerosis was not commonly seen in India

Many studies have been conducted which reports the association of optic neuritis with multiple sclerosis. Before ONTT, Jain et al [50] reported that clinical profile of optic neuritis in India is different from that of Western population. The aim of our study is to understand visual function following optic neuritis treatment in South India.

The role of corticosteroids in the management of optic neuritis was initially undertaken by Optic neuritis Treatment Trial which shaped our understanding of optic neuritis. All patients in our study received Intravenous methyl prednisolone 1 gram once daily for 3 days followed by oral steroids (1mg/kg body weight) for 2 – 6 weeks with tapering doses.

88 patients aged between 20 to 64 years (mean age 40.0) were enrolled in the study out of which 47 were male and 41 female.78 patients showed unilateral involvement, 10 patients showed bilateral involvement. A similar study was conducted by Rohit Saxena et al [48] included 83 patients between the age of 15 – 58 years (mean age 27.6 years) of which 67 cases had unilateral involvement and 16 cases had bilateral involvement. ONTT included 457 patients with ages ranging from 18 – 46 years (mean age 31.8 years) [49].

Males were commonly affected in our study (53.4%) compared to females. A similar study conducted by Jain et al[50] showed 67% of male involvement in their study. In ONTT, 77% of patients affected were females.[49]

Most common presentation in our study was defective vision present in 91.8% (90 out of 98 eyes) , headache in 12.5% (11 out of 98 eyes), dyschromatopsia in 20.4% (20 out of 98 eyes) pain on eye movements present in 29.6% (29 out of 98 eyes). Similarly Jain et al[50] found 33.3% (7 out of 42 patients) had pain on eye movements in their study. In ONTT pain was present in 93% of 295 eyes with retrobulbar

neuritis and in 90% of 162 eyes with papillitis. Majid et al[51] reported painful eye movements in 12 patients in their study.

In most of the eyes visual acuity was worse at baseline. At follow up of 1 month in 15 (19.5%) eyes visual acuity was 6/24 or better. In 45(58.4%) eyes visual acuity was 6/18- 6/6. Similarly Pedro et al reported improvement of visual acuity of 52.4% in 18 eyes out 35 eyes at follow up.[56]

Out of 98 eyes 86 (87.8%) eyes showed RAPD. 12 (12.2%) eyes were normal at baseline.

At baseline, in 15 eyes optic disc was normal, 44 eyes showed disc edema, 20 eyes showed temporal pallor, hyperemia in 20 eyes. Other findings were splinter haemorrhages seen in 2 eyes. ONTT reported swollen optic disc was seen in 35%. Nikoskelainen et al reported optic disc was normal in 46%, hyperemic/ blurred disc in 20%, disc edema in 23% and 11% showing temporal pallor.

Papillitis was seen in 65.3% and retrobulbar neuritis in 34.7 % of patients in our study. Similarly Saxena[48] et al reported 53.5% of eyes had papillitis and retrobulbar neuritis was seen in 46.5%.

Visual fields at baseline were done only in 48 eyes, it could not be performed in 50 eyes because of poor vision. Most common field defect was centrocaecal scotoma seen in 22 (22.7) eyes. 7.8% in our study had superior and inferior field defect generalised constriction of visual field and superior field loss. In our study 1 patient had altitudinal field defect at baseline which is common in NAION can also present in ON. Similarly ONTT[54] reported 48.2% showed diffuse loss , 8.3% central or centrocaecal scotoma and altitudinal field defects in 23.4% eyes which was the most common field defects at baseline. Jain et al reported concentric contraction was seen in 25% of eyes followed by central scotoma seen in 19.1% eyes.

Dyschromatopsia was recorded using Ishihara charts. At baseline 86 (87.8%)eyes it was abnormal 12(12.2%) eyes it was normal. At 1 month 53(68.8%) eyes showed improvement. 24(31.2%) did not show improvement. Jain et al reported recovery of dyschromatopsia along with recovery of vision in their study. Saxena et al[48] reported 60.6% showed

improvement in colour vision in their study. Vimala et al reported improvement of dyschromatopsia at 1 month follow up. (Mean Log mar at baseline was 9.14 which improved to 18.57)[59]

Brightness sensitivity - Out 98 eyes 83 were abnormal at baseline, in most it could not be performed due to poor vision. At follow up of 1 month 43(55.8%) eyes out of 77 eyes showed improvement. Brightness testing appeared to be more sensitive than other techniques in establishing the diagnosis.[37.]

Red desaturation – Out of 98 eyes 86 eyes were abnormal at baseline. At follow up of 1 month ,out of 77 eyes 43 eyes showed improvement. In our study we have included brightness sensitivity and Red Desaturation as one of the important parameters for evaluation of visual function. Similarly Almong et al has shown increased desaturation in ON in their study.[55]

Neuro imaging showed demyelinating lesion in the brain which was present only in 3 cases in our study showing that incidence of MS is low in South India. ONTT[54] reported that demyelinating changes

consistent with MS were seen in 37.5% (8 cases out of 32) and with contrast, it was 48.7% (203 cases out of 417). It also reported that risk of developing MS after an attack of optic neuritis was 50% and if the baseline MRI is negative the risk of MS was 25%. Saxena et al[48] reported 12 cases showed demyelinating lesion and 4 cases showed MS in their study. Jain et al reported that incidence of MS is low in southern part of India compared to northern India.

LIMITATIONS

1. The follow up period was very short to evaluate the long term prognosis.
2. Favourable visual outcome of ON prohibited patients from returning to our department for long term follow up.
3. MRI was done only in 35 patients due to financial constraints.
4. HFA was not done in all the patients due to financial constraints.

CONCLUSION

1. The age range is 20 to 64 years with mean age 40.0(12.9) years.

2. 79.6 % of patients had unilateral presentation.

3. 20.4% patients had bilateral presentation.

4. There is a slight male preponderance in our study.

5. Defective vision followed by pain on eye movements are the chief complaints in our study.

6. There was a significant improvement in visual acuity.

7. The most common presentation was papillitis (65.3%) followed by retrobulbar neuritis (34.7%)

8. Visual function like central fields, colour vision, Brightness sensitivity, Red desaturation showed a significant improvement following treatment.

9. Neuro imaging showed signs of demyelination in 3 patients.

10. 3.9% of patients were considered as atypical and requires further follow up and investigations.

11. Demyelinating disease in association with ON was less common in our study.

Visual function following optic neuritis treatment showed good recovery. There was less incidence of associated demyelination in our study.

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PROFORMA

VISUAL OUTCOME FOLLOWING OPTIC NEURITIS TREATMENT

Name _____

Age ☐ Mr.No. ☐

Gender ☐ M –Male, F- Female

Chief complaints

Onset of symptoms

Laterality 1. RE _____

2. LE _____

Defective vision RE 1. Present

LE 2. Absent

Pain on Eye Movements 1. Present

2. Absent

Headache 1. Present

2. Absent

Double Vision 1. Present

2. Absent

OCULAR EXAMINATION:

	RE	LE	1.6/6 – 6/60
BCVA	☐	☐	2.6/60 – 1/60
			3.<1/60

PUPIL

A) Direct	☐	☐	
B) Indirect	☐	☐	1. Present
C) RAPD	☐	☐	2. Absent
EOM	☐	☐	1. Full
			2. Restricted
			3. Others

CLINICAL INVESTIGATION:

	RE	LE	
Colour Vision	☐	☐	1. Normal 2. Defective
Red Color Desaturation	☐	☐	1.100% 2. <100%
Brightness Sensitivity	☐	☐	1. 100% 2. <100

FUNDUS

	RE	LE	
Normal	☐	☐	1. Yes 2. No
Disc Edema	☐	☐	1. Present 2. Absent
Hyperemia	☐	☐	
Splinter Haemorrhage	☐	☐	
Temporal Pallor	☐	☐	
Primary Optic Atrophy	☐	☐	
Secondary Optic Atrophy	☐	☐	

CENTRAL FIELDS

	RE	LE	
Normal	☐	☐	1. Yes 2. No
Enlargement of Blind Spot	☐	☐	1. Present 2. Absent
Central Scotoma	☐	☐	
Centrocaecal Scotoma	☐	☐	
Altitudinal Hemianopia	☐	☐	

NEUROLOGICAL EXAMINATION

Higher Functions	☐	1. Normal
Cranial Nerves	☐	2. Affected

Investigations

TC

DC

ESR

Mantoux

TPHA

Platelet Count

TREATMENT

Medical 1. IV Steroids

2. Oral Steroids

1st Follow Up (15 days)

	RE	LE
BCVA	☐	☐
Colour Vision	☐	☐

FUNDUS

	RE	LE	
Normal	☐	☐	1. Yes 2. No
Disc Edema	☐	☐	
Hyperemia	☐	☐	
Splinter Haemorrhage	☐	☐	1.
Present	☐	☐	
Temporal Pallor			2.
Absent	☐	☐	
Primary Optic Atrophy			
Secondary Optic Atrophy	☐	☐	

CENTRAL FIELDS

	RE	LE	
Normal	☐	☐	1. Yes 2. No
Enlargement of Blind Spot	☐	☐	
Central Scotoma	☐	☐	1. Present
Centrocaecal Scotoma	☐	☐	2. Absent
	☐	☐	

Altitudinal Hemianopia

2nd Follow Up (1 Month)

	RE	LE
BCVA	☐	☐
Colour Vision	☐	☐

FUNDUS

	RE	LE
Normal	☐	☐
Disc Edema	☐	☐
Hyperemia	☐	☐
Splinter Haemorrhage	☐	☐
Present	☐	☐
Temporal Pallor		
Absent	☐	☐
Primary Optic Atrophy		
Secondary Optic Atrophy	☐	☐

1. Yes
2. No

1.

2.

CENTRAL FIELDS

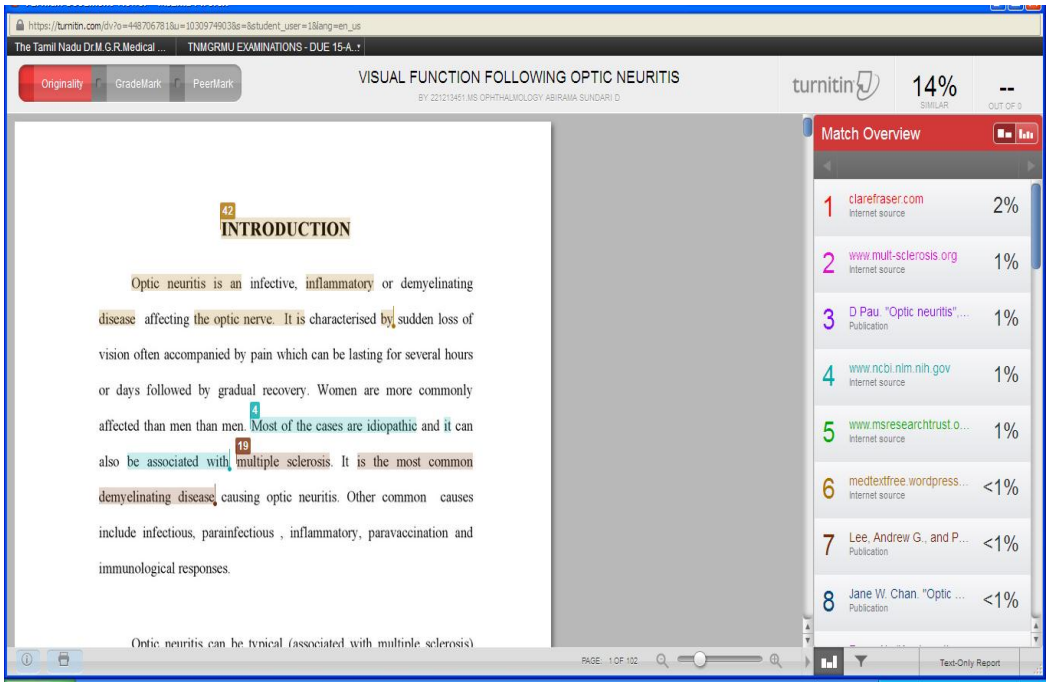
	RE	LE	
Normal	☐	☐	1. Yes 2. No
Enlargement of Blind Spot	☐	☐	
Central Scotoma	☐	☐	1. Present
Centrocaecal Scotoma	☐	☐	2. Absent
Altitudinal Hemianopia	☐	☐	

ABBREVATIONS

ON	–	Optic neuritis
MS	–	Multiple sclerosis
NFL	–	Nerve fibre layer
ONTT	–	Optic neuritis treatment trial
LONS	–	Longitudinal optic neuritis study
CHAMPS	–	Controlled High Risk Avonex Multiple Sclerosis Trial.
BENEFIT	–	Beta Interferon In Newly Emerging MS for Initial Treatment.
ETOMS	–	Early Treatment of Multiple Sclerosis Study

name	age	gender	mrno	laterality	eye	onset of symptoms	defective vision	pain on eye movements	headache	defective colour vision	bcva	bcva_new	pupil	eom	colour_vision	brightness sensitivity	red desaturation	cranial_nerves	fundus	central fields	V/A 1 month	colour vision 1 month	visual fields	fundus 1m	brightness1m	red desaturation 1m
alagu	42	female	3599729	re	1	1week	present	present	absent	absent	re1/60	1/60	RAPD	Full	poor vision	cant do	cant do	NORMAL	disc edema	cant do	6/9	normal	centrocaecal	temporal pallor	reduced	reduced
ammutha	30	female	3501647	le	2	5 days	present	absent	absent	absent	le-PL	PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	poor vision	pl	poor vision	poor vision	poa	reduced	reduced
anna mary	50	female	3398333	le	2	2 months	present	absent	absent	absent	le-pl	PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	poor vision	fcf	cant do	poor vision	mild temporal pallor	poor vision	poor vision
arockia mary	55	male	3703479	le	2	1 week	present	present	absent	absent	le-1/2/60	1/2/60	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	6/6	normal	normal	resolving disc edema	Normal	Normal
arumugam	54	male	3647974	re	1	5 days	present	absent	absent	absent	6/18p	6/18p	normal	Full	12/21	reduced	reduced	NORMAL	disc edema	centrocaecal scotoma	6/9	normal	normal	resolving disc edema	reduced	reduced
babitha	28	female	3717571	re	1	10days	absent	present	absent	absent	re-6/6p	6/6p	normal	Full	21/21	1	1	NORMAL	normal	normal	6/6	normal	normal	normal	Normal	normal
bagavathi	62	female	3505097	le	2	3 weeks	present	absent	absent	absent	le-FCF	FCF	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	poor vision	6/36	normal	centrocaecal	mild temporal pallor	Normal	normal
balasubramani	29	male	3153282	re	1	15 days	present	absent	absent	absent	be-6/18	6/18	RAPD	Full	1/21	reduced	reduced	NORMAL	normal	centrocaecal scotoma	6/6	normal	centrocaecal	normal	Normal	Normal
balasubramani			3153282	le	2		present	absent		absent		6/18	RAPD	Full	1/21	reduced	reduced	NORMAL	normal	centrocaecal scotoma	6/9	normal	centrocaecal	normal	Normal	Normal
baskar	57	male	3678489	le	2	5 days	present	absent	absent	absent	le-1/2/60	1/2/60	RAPD	Full	poor vision	cant do	cant do	NORMAL	disc hyperemia/edema	poor vision	6/9	normal	normal	normal	Normal	Normal
boomika	24	female	3525766	re	1	1 month	present	absent	absent	present	re-6/36 le-6/36	6/36	RAPD	Full	12/21	reduced	reduced	NORMAL	normal	centrocaecal scotoma	6/18	normal	poor vision	mild hyperemia	reduced	reduced
boomika			3525766	le	2		present	absent		present		6/36	RAPD	Full	12/21	reduced	reduced	NORMAL	disc hyperemia	centrocaecal scotoma	6/18	normal	normal	normal	Normal	Normal
boopathi	22	male	3508994	re	1	1week	present	present	absent	present	le-2/60	2/60	RAPD	Full	0/21	reduced	reduced	NORMAL	normal	poor vision	6/6	normal	normal	normal	Normal	Normal
chandrasekar	52	male	3465445	re	1	1week	present	absent	absent	absent	re-6/60	6/60	RAPD	Full	12/21	reduced	reduced	NORMAL	disc edema	centrocaecal scotoma	6/18	normal	normal	mild temp.pallor	reduced	reduced
chellamuthu	50	male	3551406	le	2	3days	present	absent	absent	absent	le-3/60	3/60	RAPD	Full	0/21	cant do	cant do	NORMAL	disc hyperemia/edema	cant do	6/6p	normal	normal	resolved disc edema	Normal	Normal
chellathal	50	female	3545763	re	1	20 days	present	absent	absent	present	re-1/60	1/60	RAPD	Full	cant do	cant do	cant do	NORMAL	temporal pallor	cant do	4/60	defective	sup/inf defect	pri. OA	reduced	reduced
damodaran	35	male	3347392	le	2	4days	present	absent	absent	absent	le-fcf	FCF	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	poor vision	6/12	defective	normal	temporal pallor	reduced	reduced
dhanalakshmi	27	female	3557659	le	2	20 days	present	present	absent	absent	re-fcf	FCF	RAPD	Full	cant do	cant do	cant do	NORMAL	disc hyperemia	poor vision	6/6p	normal	centrocaecal	normal	Normal	normal
ganesan	45	male	3562830	le	2	1week	present	absent	present	absent	le-4/60	4/60	RAPD	Full	1/10	defective	defective	NORMAL	disc edema	not done	6/12	normal	normal	temporal pallor	Normal	Normal
gangadaram	35	male	3657932	le	2	10 days	present	absent	absent	absent	le-5/60	5/60	RAPD	Full	2/21	reduced	reduced	NORMAL	disc edema	centrocaecal scotoma	6/24	normal	normal	resolving disc edema	Normal	Normal
gokila	21	female	3568642	re	1	2days	present	absent	absent	present	re-pl	PL	RAPD	Full	poor vision	reduced	<100%	NORMAL	disc hyperemia	poor vision	6/6p	normal	normal	normal	Normal	Normal
gokilavani	36	female	3721671	le	2	3days	absent	present	absent	absent	le- 6/6p	6/6p	normal	Full	13/21	reduced	>50%	NORMAL	normal	centrocaecal scotoma	6/6	normal	normal	normal	Normal	Normal
gunavathi	22	female	3627579	le	2	4days	absent	present	absent	present	le-6/6	6/6	normal	Full	21/21	1	1	NORMAL	normal	normal						
iyyammal	50	female	3624310	le	2	10 days	present	absent	absent	absent	le-hm	HM	RAPD	Full	poor vision	cant do	cant do	NORMAL	disc edema	poor vision	2/60	cant do	poor vision	hyperemia d.edema	poor vision	poor vision
jeganath	35	male	3684806	le	2	2 days	absent	present	absent	absent	le-6/6	6/6	RAPD	Full	21/21	1	1	NORMAL	disc edema	normal						
juliet	39	female	3633617	le	2	1month	present	absent	absent	absent	le-6/36	6/36	RAPD	Full	21/21	1	1	NORMAL	disc edema	normal	6/24p	normal	normal	normal	Normal	Normal
kalaivanan	37	male	3685404	le	2	1 day	present	absent	absent	absent		NOPL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc hyperemia/edema	poor vision	6/36	normal	centrocaecal	resolving disc edema	reduced	reduced
kalaivanan			3685404	re	1		present	absent		absent	be-pl+ no pl	PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc hyperemia/edema	poor vision	6/36	defective	centrocaecal	resolving disc edema	reduced	reduced
kalyani	40	male	3701843	le	2	6days	present	absent	absent	absent	le- 3/60	3/60	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	centrocaecal scotoma	6/12	normal	normal	resolving disc edema	Normal	Normal
kamalahasan	33	male	3647584	re	1	10 days	present	absent	absent	absent	re-1/60	1/60	RAPD	Full	0/21	cant do	cant do	NORMAL	disc hyperemia	poor vision						
karmegam	60	male	3533665	le	2	5days	present	absent	absent	present	re-4/60 le-fcf	4/60	RAPD	Full	poor vision	defective	nil	NORMAL	disc edema	Poor vision	1/60	not done	diffuse constriction	temporal pallor	reduced	reduced
karuppasamy	27	male	3477145	le	2	10 days	present	present	absent	present	le-5/60	5/60	RAPD	Full	0/21	reduced	<100%	NORMAL	disc edema	poor vision	6/6p	normal	normal	resolving disc edema	Normal	Normal
karuppiah	64	male	3418305	le	2	20 days	present	present	present	absent	le-1/60	1/60	RAPD	Full	poor vision	poor vision	0.3	NORMAL	disc edema	centrocaecal scotoma	6/9	normal	normal	mild hyperemia	Normal	Normal
leelavathi	43	female	3614240	re	1	10 days	present	absent	absent	absent	re-pl	PL	RAPD	Full	poor vision	cant do	cant do	NORMAL	temporal pallor	poor vision	hm	poor vision	poor vision	mild temporal pallor	poor vision	poor vision
mahabu nisha	32	male	3695795	re	1	15 days	present	absent	present	absent	be-6/12	6/12	normal	Full	16/21	1	1	NORMAL	disc edema	normal	6/6	normal	normal	temporal pallor	Normal	Normal
mahabu nisha			3695795	le	2		present	absent		absent		6/12	normal	Full	13/21	1	1	NORMAL	disc edema	normal	6/6	normal	normal	resolving disc edema	Normal	Normal
malaiammal	62	female	3706114	le	2	4 days	present	absent		absent		HM	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	Poor vision	6/18	normal	centrocaecal	temporal pallor	reduced	reduced
malaiammal			3706114	re	1		present	absent	absent	absent	re-6/60 le-hm	6/60	RAPD	Full	12/21	poor vision	poor vision	NORMAL	temporal pallor	enlargement of blind spot	6/12	normal	arcuate scotoma	temporal pallor	reduced	reduced
mallika	50	male	3697246	le	2	6 days	present	absent	absent	absent	le-pl	PL	normal	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	6/24	normal	normal	temporal pallor	Normal	Normal
manjula	50	female	3522601	re	1	4 days	present	absent	absent	present	re-6/60	6/60	RAPD	Full	9/21	reduced	reduced	NORMAL	disc hyperemia	poor vision	6/6	normal	normal	resolved disc edema	Normal	Normal
mariammal	55	female	3532296	le	2	3days	present	absent	absent	present	le-pl	PL	RAPD	Full	poor vision	poor vision	defective	NORMAL	temporal pallor	poor vision	6/24	normal	centrocaecal	temporal pallor	Normal	Normal
mariammal	50	female	3560236	re	1	2weeks	present	present	absent	absent	re-5/60	5/60	RAPD	Full	13/21	reduced	reduced	NORMAL	temporal pallor	centrocaecal scotoma	6/24p	normal	centrocaecal	temporal pallor	reduced	reduced
meenakshi	40	female	3625372	le	2	1week	present	absent	present	absent	re-HM	HM	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	poor vision						
meenal	31	female	3707447	le	2	2 weeks	absent	present	absent	absent	le-6/6p	6/6p	normal	Full	21/21	1	1	NORMAL	normal	temporal field defect						
moshe	45	male	3678815	re	1	20 days	present	absent	absent	absent	re-pl	PL	RAPD	Full	poor vision	cant do	cant do	NORMAL	temporal pallor	poor vision	pl	poor vision	poor vision	gross t.pallor	poor vision	poor vision
murugan	30	male	3287750	re	1	20days	present	absent	absent	absent	re-1/60	1/60	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	cant do	6/6p	normal	normal	mild hyperemia	Normal	Normal
murugan.s	50	male	3380620	le	2	1week	present	absent	absent	absent	le-6/18p	6/18p	RAPD	Full	14/21	reduced	reduced	NORMAL	hyperemia disc edema	centrocaecal scotoma	6/60	defective	altitudinal	mild hyperemia re.d .edema	reduced	reduced
muthuirulayee	26	female	3647927	re	1	2 days	present	absent	absent	present	re-5/60	5/60	RAPD	Full	1/21	cant do	cant do	NORMAL	disc edema	centrocaecal scotoma						
muthupetchi	60	female	3590767	re	1	1 month	present	present	absent	absent	re-1/60	1/60	RAPD	Full	cant do	cant do	cant do	NORMAL	temporal pallor	cant do	1/60	defective	poor vision	temporal pallor	reduced	reduced
nagalingam	35	male	3626116	le	2	10 days	present	absent	absent	present	le-2/60	2/60	RAPD	Full	poor vision	cant do	reduced	NORMAL	disc hyperemia	cant do						
nagaraj	28	male	3530513	le	2	3days	absent	present	absent	present	le-6/6	6/6	RAPD	Full	14/21	reduced	reduced	NORMAL	normal	centrocaecal scotoma	6/6p	normal	normal	normal	Normal	Normal
nithya	30	male	3683413	le	2	15 days	present	absent	absent	absent	le-3/60	3/60	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	poor vision						
padmavathy	33	female	3393963	le	2	1month	present	absent	absent	present	le-PL	PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	6/36	normal	normal	resolving disc edema	Normal	Normal
palaniammal	42	female	3629294	re	1	4 days	present	absent	absent	absent	be-1/60	1/60	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	enlargement of blind spot	6/18	normal	normal	mild hyperemia resol d.edema	Normal	Normal
palaniammal			3629294	le	2		present	absent		absent		1/60	RAPD	Full	cant do	cant do	cant do	NORMAL	disc edema	enlargement of blind spot	6/24	normal	normal	temporal pallor	Normal	Normal
pandiammal	42	female	3408493	re	1	1month	present	present	absent	present	le-2/60	2/60	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	normal	centrocaecal scotoma	6/12	normal	normal	normal	Normal	Normal
parameswari	50	female	3462579	re	1	1week	present	present	absent	absent	re-fcf le-2/60	FCF	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	generalized constriction	6/24	normal	sup/inf defect	early temporal pallor	reduced	reduced
parameswari			3462579	le	2		present					2/60	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	temporal pallor	Poor vision	6/9	normal	sup/inf defect	early temporal pallor	reduced	reduced
paul ananth	44	male	3607237	le	2	2 days	present	present	absent	absent	le-6/18p	6/18p	RAPD	Full	14/21	reduced	reduced	NORMAL	normal	normal						
periyakamatchi	64	male	3565711	le	2	4days	present	absent	absent	present	pl	PL	RAPD	Full	cant do	cant do	poor vision	NORMAL	temporal pallor	poor vision	1/60	defective	poor vision	early t.pallor	poor vision	poor vision
ponnusamy	20	male	3561080	re	1	20 days	present	absent	absent	absent	re-pl	PL	RAPD	Full	cant do	cant do	poor vision	NORMAL	temporal pallor	poor vision	pl	defective	poor vision	pri. OA	poor vision	poorvision
rabika banu	25	female	3623624	re	1	4days	absent	present	absent	absent	re-6/6p	6/6p	normal	Full	21/21	1	1	NORMAL	disc hyperemia/edema	normal	6/6	normal	normal	resolving disc edema	Normal	Normal
rajasekaran	54	male	3499171	le	2	3 days	present	present		absent		2/60	RAPD	Full	poor vision	cant do	poor vision	NORMAL	disc edema	Superior field loss						
rajasekaran			3499171	re	1		present	present	present	absent	re-1/60le-2/60	1/60	RAPD	Full	poor vision	cant do	poor vision	NORMAL	disc edema	Paracentral scotoma						
rajeswari	22	female	3624278	re	1	1week	present	absent	absent	absent	re-4/60	4/60	normal	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision						
raman	45	male	3541113	le	2	4days	present	absent	absent		le-fcf	FCF	RAPD	Full	poor vision	defective	poor vision	NORMAL	temporal pallor	poor vision	6/6p	normal	poor vision	early temporal pallor	Normal	Normal
ramawsamy	24	male	3714091	re	1	2 days	present	absent	absent	absent	re-4/60	4/60	RAPD	Full	0/21	reduced	poor vision	NORMAL	disc edema	centrocaecal scotoma						
ramuthai	64	female	3401322	re	1	3months	present	absent	absent	absent	le-pl	PL	RAPD	Full	cant do	cant do	poor vision	NORMAL	temporal pallor	poor vision	5/60	defective	central scotoma	mild temporal pallor	reduced	reduced
revathy			3517695	le	2		present	absent		absent		FCF	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	6/6	normal		mild hyperemic resol d.edema	Normal	Normal
revathy	29	female	3517695	re	1	1 week	present	absent	absent	absent	RE-FCF LE FCP	FCF	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	6/6	normal	normal	resolved disc edema	Normal	Normal
rober rajkumar	50	male	3446034	le	2	2days	present	present	absent	present	le-6/36	6/36	RAPD	Full		reduced	reduced	NORMAL	disc hyperemia	centrocaecal scotoma	6/9	normal	normal	mild hyperemia	Normal	Normal
samuel	24	male	3689941	re	1	10 days	present	present	absent	absent	re-6/9p	6/9p	RAPD	Full	1/21	reduced	reduced	NORMAL	disc edema	enlargement of blind spot						

sangeetha	30	female	3483052	le	2	1month	present	present	present	absent	le-6/6	6/6	RAPD	Full	21/21	1	1	NORMAL	disc edema	centrocaecal scotoma	1/2/60	poor vision	poor vision	temporal pallor	poor vision	poor vision
sangumuthu	42	male	3696406	le	2	4 days	present	present	absent	absent	le-1/60	1/60	RAPD	Full	poor vision	cant do	cant do	NORMAL	normal	normal	6/6	normal	normal	normal	Normal	Normal
saravani	23	female	3637929	re	1	3 days	present	absent	present	absent	re-1/2/60	1/2/60	normal	Full	0/21	poor vision	poor vision	NORMAL	disc edema	poor vision						
sathish	29	male	3582982	le	2	20 days	present	absent	absent	absent	le-hm	HM	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc hyperemia	poor vision	6/9	normal	centrocaecal	hyperemia	Normal	Normal
sathiya rama	60	female	3652178	le	2	20 days	present	absent	absent	absent	le-pl	PL	RAPD	Full	poor vision	poor vision	cant do	NORMAL	temporal pallor mild edema	poor vision	1/2/60	cant do	poor vision	temporal pallor	reduced	reduced
satinder singh	25	male	3650563	le	2	2weeks	present	absent	absent	absent	le-6/60	6/60	RAPD	Full	2/21	reduced	reduced	NORMAL	disc edema	inferior and sup field defect	6/6p	normal	normal	normal	Normal	Normal
selvam	60	male	3700221	re	1	10 days	present	absent	present	absent	re-pl+	PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision	3/60	poor vision	central scotoma	normal	reduced	reduced
shanmugam	41	male	3429775	le	2	15 days	present	present	absent	present	le-6/24	6/24	RAPD	Full	21/21	reduced	reduced	NORMAL	disc hyperemia	normal	6/9	normal	normal	normal	Normal	Normal
sivakumar	41	male	3716211	re	1	13days	present	absent	absent	absent	re- 6/60	6/60	RAPD	Full	3/21	reduced	reduced	NORMAL	disc edema	centrocaecal scotoma						
sulaika	45	female	3710301	le	2	3 weeks	present	absent	absent	absent	le-hm	HM	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	disc edema	poor vision						
surendar	22	male	3600660	le	2	7 days	present	absent	absent	absent	le-5/60	5/60	RAPD	Full	1/21	reduced	reduced	NORMAL	disc hyperemia/edema	defective	6/12	normal	poor vision	resolved disc edema	reduced	reduced
suresh	22	male	3513263	le	2	3days	present	present	absent	present	le-1/60	1/60	RAPD	Full	0/21	poor vision	poor vision	NORMAL	disc edema	generalised	6/6	normal	normal	resolving disc edema	Normal	Normal
tamilarasi	32	female	2782001	re	1	2 days	present	absent	absent	absent	re-fcf	FCF	RAPD	Full	poor vision	cant do	cant do	NORMAL	disc edema	poor vision	pl	poor vision	poor vision	hyperemia d.edema	reduced	reduced
teena paul	42	female	3521274	re	1	3 weeks	present	present	absent	absent	re- 6/6	6/6	normal	Full	21/21	1	1	NORMAL	normal	centrocaecal scotoma	6/6	normal	normal	normal	Normal	Normal
umadevi	36	female	3281551	re	1	20 days	present	absent	absent	absent	re-3/60	3/60	RAPD	Full	1/21	reduced	poor vision	NORMAL	disc edema	poor vision						
vanaja	45	female	3621081	le	2	10 days	present	absent	absent	absent	le-6/18p	6/18p	RAPD	Full	0/21	reduced	reduced	NORMAL	temporal pallor	centrocaecal scotoma						
veeranan	62	male	3559839	re	1	1week	present	absent	absent	absent	re-6/18	6/18	RAPD	Full	21/21	1	1	NORMAL	normal	centrocaecal scotoma	6/9	normal	centrocaecal	normal	Normal	Normal
veeravel	61	male	3631863	le	2	4days	present	present	absent	absent	le-6/36	6/36	RAPD	Full	0/21	reduced	reduced	NORMAL	normal	peripheral loss	6/12	normal	poor vision	normal	Normal	Normal
vellaiyammal	55	female	3628515	le	2	1 month	present	present	present	absent	le-4/60	4/60	RAPD	Full	0/21	cant do	poor vision	NORMAL	disc edema	altitudinal field defect	4/60	poor vision	poor vision	mild temporal pallor	reduced	reduced
velmurugan	30	male	3562834	le	2	2days	absent	present	absent	absent	le-6/36	6/36	RAPD	Full	21/21	1	1	NORMAL	disc edema	normal	6/6p	normal	normal	normal	Normal	Normal
velu	40	male	3568511	re	1	1week	present	absent	absent	present	re-6/60	6/60	RAPD	Full	10/21	reduced	reduced	NORMAL	disc edema	Paracentral scotoma	6/12	normal	centrocaecal	mild temporal pallor	Normal	Normal
veni	30	female	3557795	re	1	2 days	present	absent	absent	absent	re-hm	HM	RAPD	Full	cant do	poor vision	poor vision	NORMAL	disc edema	poor vision	1/2/60	defective	poor vision	mild temporal pallor	reduced	reduced
venketeswari	28	female	3714825	le	2	7 days	present	absent	present	absent	le-1/2/60	1/2/60	RAPD	Full	0/21	reduced	poor vision	NORMAL	disc edema	poor vision						
vijayan	35	male	3573475	le	2	20days	present	absent	absent	absent	le-1/60	1/60	RAPD	Full	0/21	reduced	poor vision	NORMAL	temporal pallor	poor vision	6/60	defective	sup/inf defect	mild temporal pallor	reduced	reduced
yasmin	25	male	3511227	re	1	5days	present	absent	present	absent	re-3/60 le-pl	3/60	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	hyperemic disc edema	Central scotoma	6/60	defective	central	resolving disc edema	reduced	reduced
yasmin			3511227	le	2		present	absent		absent		PL	RAPD	Full	poor vision	poor vision	poor vision	NORMAL	hyperemia/ disc edema	Poor vision	6/24	normal	centrocaecal	mild hyperemia	reduced	reduced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INTRODUCTION
Optic neuritis is an infective, inflammatory or demyelinating disease affecting the optic nerve. It is characterised by sudden loss of vision often accompanied by pain which can be lasting for several hours or days followed by gradual recovery. Women are more commonly affected than men than men. Most of the cases are idiopathic and it can also be associated with multiple sclerosis. It is the most common demyelinating disease causing optic neuritis. Other common causes include infectious, parainfectious , inflammatory, paravaccination and immunological responses.
Optic neuritis can be typical (associated with multiple sclerosis)
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INTRODUCTION